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Volume 20 October, 1937 Number 10

HYPERTENSIVE HEART DISEASE—ITS CLINICAL-PATHOLOGICAL MANIFESTATIONS*

FRANCIS D. MURPHY, M.D., ROBERT M. WOODS, M.D., and JOHN GRILL, M.D.
Milwaukee, Wisconsin

THE term hypertensive heart disease occupies a unique position in the modern classification of diseases of the heart. Although it is by far the commonest type of heart disease it is the least understood of any. It has been recognized ever since Bright,[6] in one of his original papers, pointed out that cardiac hypertrophy played a part along with kidney sclerosis and hypertension in Bright's disease. Yet the true significance of this cardiac hypertrophy has only recently begun to be recognized. Perhaps the term may be looked upon only as a new name for an old condition, but it is more than that. This name places the emphasis upon the features of the disease that should be emphasized, and introduces a new point of view in the interpretation of the clinical features. It tends to identify a kind of heart disease that has lacked identification. It is hoped that the natural sequelæ of this correct delineation will be its earlier diagnosis, a better understanding of its course, and more effective methods of treatment.

In former years the close association of cardiac hypertrophy, hypertension, and renal sclerosis led observers to evaluate the symptoms of hypertensive heart disease in terms of renal insufficiency and failure. So well were the lessons of kidney pathology learned that for over three-quarters of a century after Bright the hypertrophic heart and its behavior were overshadowed by the study of the shrunken, contracted, sclerotic kidney. Albuminuria, hypertension, and cardiac enlargement came to be looked upon as satisfactory evidence for a diagnosis of chronic interstitial nephritis; and the patient with these symptoms was watched zealously for the oncoming renal failure and uremia which frequently never occurred.

Hypertensive heart disease differs from other cardiac disorders in that several etiological factors are closely interwoven in its development. While it is true that it is the result of the combined action of hypertension and arteriosclerosis, it is not a disease which lacks unity. The distinctive pathological lesions in addition to the characteristic symptoms and clinical course are sufficient for classing it as a separate form of heart disease. The modern concept of hypertensive heart disease, then, may be given as follows: The heart, which has been overstrained by persistent arterial hypertension, hypertrophies, becomes impoverished by lack of proper nutrition due to coronary arteriosclerosis, and fails as a result of a combination of these two forces.

The upward trend in the mortality rate from heart disease in general has attracted widespread attention in the profession and among the laity. Explanations given by the Bolduans,[5] Cohn,[10] Hedley,[21] Dublin and Armstrong,[12] and others[13] imply that the great increase in the mortality rate in heart disease is more apparent than real. In spite of this, the problem of the management of the individual patients is not minimized. Undoubtedly in modern times more people grow older because diseases such as tuberculosis, diabetes, typhoid fever, and the infectious diseases have been better controlled. As a result people live to middle age or older into the period

* From the Department of Medicine, Marquette University, and the Department of Medicine, Milwaukee County Hospital. Presented at the annual meeting of the Minnesota State Medical Association, St. Paul, Minnesota, May 4, 1937.

TABLE I. DISTRIBUTION OF DEATHS IN THE UNITED STATES IN 1930, 1932, AND 1933 ACCORDING TO ABRIDGED INTERNATIONAL LISTS OF CAUSES OF DEATH

Cause of Death	Number			Rate per 100,000 estimated population			Per Cent		
	1933	1932	1930	1933	1932	1930	1933	1932	1930
Diseases of the heart	286,360	268,696	253,084	227.8	223.7	213.5	21.3	20.5	18.8
Cancer and malignant tumors	128,479	122,739	115,265	102.2	102.2	97.2	9.6	9.4	8.6
Nephritis	104,264	104,571	107,619	83.0	87.2	90.8	7.8	8.0	8.0
Violent and accidental (except homicide and suicide)	91,087	85,868	95,527	72.5	71.5	80.6	6.8	6.6	7.1
Pneumonia	86,949	92,474	98,657	69.6	77.0	83.2	6.5	7.1	7.3
Tuberculosis of the respiratory system	67,422	67,789	75,120	53.6	56.4	63.4	5.0	5.2	5.6

of life when heart disease due to degenerative changes becomes more frequent. The importance of heart disease in mortality rate is nowhere better shown than in the United States Department of Commerce, Bureau of Census, Mortality Statistics.[44] Here it is seen that in the year 1933, there were 286,360 deaths due to diseases of the heart, as compared to 128,479 due to cancer and malignant tumors and 86,949 to pneumonia (Table I). When we turn, however, to vital statistics in order to find how common hypertensive heart disease is we are keenly disappointed. Inaccurate, misleading, and obsolete diagnostic terms are, to some extent at least, responsible for the lack of identification of hypertensive heart disease. The apparent unimportance of hypertensive heart disease shown in vital statistics is misleading. Cabot[7] states that it is commoner than any other type of cardiac malady and is more common than all other forms of heart disease put together. Hedley[20] reports that 61.4 per cent of the total cardiac deaths in Washington, D. C., during 1932, were due to arterial hypertension and arteriosclerosis.

Confusion Due to the Inaccurate Use of Terms

Terminology and classifications have considerable influence over our ideas of the nature of diseases. Precise terms reflect our understanding of disease and from time to time should be modified to show our changing concepts. The accurate use of medical terms and clear-cut classifications become valuable aids in the understanding of diseases while inaccurate and uncertain use of them is misleading and tends to cause confusion. A lack of understanding of the true nature of hypertensive heart disease may to no small degree be attributed to a confusion in names. This common heart condition in which the kidneys, arteries, and heart may all contribute to the clinical picture has been given a variety of names. Frequently the term implied that the kidney damage was the important phase, as when such a term as "chronic interstitial nephritis" was used to identify the disease. Others saw the arteriosclerosis of the coronaries as the chief disorder and applied the term "arteriosclerotic heart disease." A source of vagueness was occasioned by the introduction of terms to depict the fibrous tissue growth in the myocardium. "Arteriosclerotic heart disease," "myofibrosis," "fibroid disease of the heart," "fibrous myocarditis," and "chronic myocarditis" were used to designate this condition. The term chronic myocarditis, introduced by Weigert,[45] has been the one most extensively used and is the most misleading. This diagnosis often covers up lack of understanding of the genuine nature of the disease. It is a diagnostic term of convenience. Its use should be restricted to designate genuine chronic myocarditis. As Willius[48] points out, in the majority of instances in which a diagnosis of chronic myocarditis is rendered, the patient has either coronary sclerosis or hypertensive heart disease or both. In reality, he points out, infection and inflammation have no part whatsoever in the resulting phenomena.

Histological examination of the heart muscle led to an erroneous interpretation of the fibrous tissue present. The patchy fibrosis and the cellular infiltration may be explained as the result of occlusion of branches of the coronary arteries. Genuine chronic myocarditis may follow certain infectious diseases such as diphtheria,

rheumatic fever and others, but this term has come into general use as a description for the form of heart disease that occurs with hypertension and coronary arteriosclerosis. Not only has this term been used to identify the pathological lesions of different pathogenesis but it has been applied to designate clinical myocardial failure as well. Pardonable confusion has been added by the fact that hypertensive patients are frequently seen by the physician after heart failure has become established and the blood pressure has fallen to almost normal levels. A post mortem examination reveals advanced coronary sclerosis, a hypertrophic heart, and renal arteriosclerosis. These pathological lesions are the identification marks of a former persistent hypertension. Chronic myocarditis, then, is a rare disease, but the name is one that is erroneously applied with great frequency to the hypertrophic heart.

Dilatation and hypertrophy of the left ventricle in cases of hypertensive heart disease lead at times to stretching of the mitral ring and this is diagnosed clinically by a soft systolic mitral murmur. Mitral insufficiency is the diagnosis sometimes used for such cases, but this is wrong. Genuine mitral insufficiency is not present in the sense that there is organic valvular disease, but it is so-called relative mitral insufficiency.

Although various authors had recognized the importance of cardiac hypertrophy and failure as a sequel to hypertension in Bright's disease it was Janeway[28] who first clearly identified this condition. From an analysis of 458 cases of hypertension he stated that in the largest group the clinical picture was that of some degree of cardiac insufficiency and that the patients most often died of cardiac failure. He designated these cases as "hypertensive cardiovascular disease" because the most prominent symptoms associated with the high blood pressure were cardiac rather than renal. More recently Fahr[14] has aided in the proper delineation of this disease and has popularized the term "hypertensive heart disease."

Hypertension, Cardiac Hypertrophy and Coronary Sclerosis

Hypertension.—Usually the hypertension itself in the syndrome of hypertensive heart disease is looked upon as the most important feature. Notwithstanding the fact that the hypertension is an important factor in the bringing on of cardiac dilatation and hypertrophy it is widely acknowledged that the hypertension is not the only element in the production of heart failure. The arteriosclerosis which goes hand in hand with hypertension is recognized more and more as an important component in this disease. The respective parts played by these two disorders have not been clearly defined as yet. Fahr[14] believes that the term hypertensive heart disease should connote both conditions.

Certain impressions in regard to the etiology of hypertension have emerged from clinical and experimental investigation during recent years. Earlier writers (Bright,[6] Traube[42] and Johnson[29]) believed that hypertension and cardiac hypertrophy were the direct outcome of renal disease. Later on Gull and Sutton,[18] Huchard,[27] Mahomed,[33] and Allbutt[1] were pioneers in placing emphasis on the dissociation of hypertension and cardiac hypertrophy from renal sclerosis. Following the widespread use of the sphygmomanometer in medicine, hypertension was diagnosed much earlier than previously and the relationship between the hypertension, renal disease and cardiac hypertrophy took on a different aspect. The prominence of symptoms relative to the heart became more widely recognized, and as a sequel to this there was a gradual but determined shift of emphasis from the renal complications of hypertension to the cardiac manifestations. Janeway[28] observed that gradual heart failure occurred in 50 per cent of hypertensive patients, and therefore the safeguarding of the heart is the main therapeutic indication.

It is generally accepted that in hypertension there is an increase of peripheral resistance, but the cause of this is not so widely agreed upon. The former theory was that arteriosclerosis, a well recognized organic change in the peripheral arteries and arterioles, was the cause. Now it is believed that a vasoconstriction exists and that the real cause of hypertension is a functional rather than an organic change in the vascular system. Recently Prinzmetal and Wilson,[37] after a series of blood flow experiments, concluded that a vascular hypertonus exists independent of the vasomotor nerves and that this intrinsic vascular hypertonus is widely spread throughout the vascular system. They believe that the vasomotor activity which is present is merely an im-

posed factor and not the chief one in causing hypertension. Pickering,[36] too, has come to substantially the same conclusion after conducting almost identical experiments.

Following experimental work on dogs, Goldblatt, Gross and Hanzal[17] produced evidence which tends to confute the theory that splanchnic nerve efferent impulses exert a significant control on the vasomotor mechanism and that their removal does not permanently reduce hypertension. They excised bilaterally the entire thoracic portion of the splanchnic nerve and the lower four dorsal sympathetic ganglia. This operation failed to prevent the development of hypertension following the partial clamping of the renal arteries. In another set of experiments they found that splanchnicectomy failed to lower the blood pressure permanently after it had been persistent for about four years following the clamping of the renal arteries. Without controverting the clinical reports of the beneficial effects of splanchnicectomy they do not believe such operations are justifiable in the treatment of hypertension.

One is led to believe from the vast amount of experimental and clinical work done that in hypertension there is an intrinsic disorder of the vascular tissue and that these very early so-called functional changes are not detectable by microscopic examination. The conclusions reached by experimenters in the field of hypertension substantiate in a way impressions held by numerous clinicians for a long time, that in hypertension there is fundamentally an intrinsic disorder of the muscle and elastic tissues of the arteries which is primarily responsible for the hypertension. Undoubtedly the sympathetic nervous influence plays a part, but most authorities agree that it is a minor factor.

Many things have been considered as possible etiological agents in hypertension. It has become almost traditional to attribute this disorder to some fault in our modern mode of living. Tea, coffee, alcohol, meat, and tobacco have from time to time been accused of being instrumental in its production, and some have called the pace of modern life an important etiological factor. The influence of such things in the etiology of arteriosclerosis and hypertension is questionable when one considers the paleopathological studies of Marc Armand Ruffer,[38] who says that the cause of arteriosclerosis three thousand years ago is as obscure as it is in modern people. Tobacco can be eliminated because this drug was not used in ancient Egypt. Alcohol played a part in the Egyptian social life. Beer was common and wine was made and imported into the country, but Ruffer believes that the Egyptians were not a race of drunkards. His experiences during the Mussulman pilgrimage are also interesting. At that time he made over 800 post mortem examinations of these people who had never touched alcohol and found that diseases of the arteries were as common as in people who take alcohol regularly. Furthermore, he believes that the theory of the wear and tear of modern life is a myth, the fact being that our life is easier and that we work less than did our ancestors. One may assume, then, that arteriosclerosis, which may be looked on as the tombstone of hypertension, was as prevalent three thousand years ago as it is today and that its etiology was just as obscure.

From the vast amount of experimental work, statistical and clinical studies, one observes that there are many possible factors that may play a part in the etiology of hypertension and arteriosclerosis. Furthermore, this lack of exact knowledge concerning hypertension must not be taken to mean that nothing is known about this condition. From the practical aspect much important and desirable information has been accumulated from years of clinical study. As an example it has been found that in the etiology there are two uniform and constant elements which are of outstanding importance, the familial tendency and the age factor.

Cardiac Hypertrophy.—An enlarged heart is a diseased one that tends to fail. When the muscle of the left ventricle is subjected to persistent strain, as occurs in chronic hypertension, dilatation and hypertrophy take place. Both are responses of heart muscle fibers to increased work. The exact cause of hypertrophy of the individual muscle fiber is unknown. In the absence of valvular heart disease or pericardial adhesions an enlarged hypertrophic heart may be looked upon as evidence that hypertension is present or has been in the past. Under the strain the muscle fiber elongates, resulting in cardiac dilatation. This in turn leads to a thickening of the fiber in order to restore the length-breadth ratio that prevailed in normal conditions. Christian[8] and others contend that cardiac dilata-

tion and hypertrophy are not advantageous compensatory changes but are for the most part a sign of intrinsic muscle fiber weakness; they hold that such changes are not normal responses to effort and are always undesirable effects.

Many authorities agree that hypertension is the most important factor in the production of cardiac hypertrophy and that prolonged muscular exertion produces muscle hypertrophy. Since the left ventricle is called upon to perform more work in chronic hypertension, hypertrophy of that chamber is not to be wondered at. Deutch and Kauf[11] and Herxheimer[24] have shown that strenuous exercise produces genuine hypertrophy of the heart. Tung and his associates[43] in a study of a number of ricksha pullers, found that twenty-one or 45 per cent of forty-six pullers showed definite cardiac enlargement, and concluded that chronic exertion may cause hypertrophy which is considered to be a physiological response but is no indication of disease. Studies in the physiology of exercise of growing dogs were made by Steinhaus, Hoyt, and Rice.[40] They concluded that a work hypertrophy in the heart is produced by exercise such as swimming and running. The right and left ventricles both are involved with excessive hypertrophy in favor of the left heart. It is easy to visualize that the left ventricular chamber bears the brunt of the load in hypertension and is more apt to fail than the right chamber.

In a study of myocardial changes in hypertension, Levine[31] examined twenty-seven hearts. Six had marked sclerosis of the large coronaries, six had sclerosis of the arterioles, eleven had marked organic changes in the arterial tree, and sixteen had changes too slight to have seriously interfered with the blood supply. In all cases there were myocardial scars, but the scarring was more marked in the hearts with more advanced arteriosclerosis. He states that in hypertension there is cardiac hypertrophy, but agreement has not been reached as to what other changes may develop. As Levine points out, coronary sclerosis in some hypertensive hearts seems sufficient to explain the myocardial damage, while in others it does not, and therefore some other explanation for these changes must be given. He believes that there is a functional spasm of the terminal arterioles followed by changes which finally lead to scarring. The anatomical changes in the myocardium and myocardial failure resulting from hypertension were studied by Clawson.[9] He points out that myocardial fibrosis and coronary sclerosis are the chief changes. Myocardial strain, he emphasizes, is not a cause of myocardial fibrosis, and many cases of myocardial failure show no changes in the heart muscle.

Aside from valvular heart disease, pericarditis, and hypertension there are occasional instances of so-called idiopathic cardiac hypertrophy. If hypertensive heart disease and failure could be explained by assuming that the increased burden of hypertension was the cause, and cardiac dilatation and hypertrophy the result, the matter would be greatly simplified, but this problem is much more intricate. There is a hypertrophic heart to which is added fibrosis and coronary arteriosclerosis. As most observers are unwilling to believe that work hypertrophy is a cause of subsequent failure other explanations must be sought. The cardiac hypertrophy may be looked on then as a cardinal sign of hypertension. The hypertensive heart may be taken to mean a hypertrophied heart and often an incompetent one without pericarditis or valvular lesion.

Coronary Sclerosis.—Hypertensive heart disease may last for years without causing any discomfort or leading to limitation of the patient's activity. Frequently this condition is discovered on a life insurance examination or some other routine. Just why the hypertensive heart fails is not exactly known, but it is believed that at least three factors contribute to this failure: (a) the hypertension itself with its persistent burden upon the left ventricle, (b) the impoverishment of the heart muscle, which is due to coronary arteriosclerosis with partial occlusion, (c) reduced oxygen diffusion of the heart muscle fibers due to thickness of the heart wall. Fahr's[15] observations led him to believe that coronary sclerosis is found in 90 per cent of all patients dying with hypertension, and Bell[3] found 75 per cent of cases of coronary sclerosis associated with hypertension. Averbuck,[2] too, emphasized that heart failure in hypertension is most commonly associated with coronary sclerosis and thrombosis. In his series of forty cases he found 34, or 85 per cent, in the cardiac group had significant coronary arterial involvement, whereas only three, or 10 per cent, of the cases of the control group had significant artery changes. Yet there

were six, or 15 per cent, of the forty cases in which no such organic changes in either the coronary arteries or heart muscle were present to account for the cardiac failure.

Levine[30] points out that the exact rôle of hypertension in hypertensive heart disease is not clear. There is a group of cases having hypertension and angina, he states, in which the blood pressure falls after an attack of coronary disease. Neither the intensity nor the duration of hypertension can entirely account for the development of heart failure. The coronary arteries seem to be the determining factor in many cases, but there are instances in which the gross appearance of the coronaries is normal and yet the heart fails. In a series of one hundred cases studied clinically and post mortem, Scott[39] found that myocardial failure was the cause of death in 68 per cent of them. Coronary sclerosis, he states, occurred frequently, but he gives no exact figures.

Of 155 hypertensive patients studied by Nuzum and his associates,[35] there was clinically detectable coronary arteriosclerosis in 44.5 per cent of the cases. Sixty-five patients came to autopsy and a clinical diagnosis of coronary sclerosis was confirmed in 97 per cent.

Lisa[32] observed advanced changes in the heart wall in all of a series of ten cases of hypertensive heart disease with failure. Marked hypertrophy was an outstanding feature. Many microscopic necrotic foci associated with fragmentations and areas of wavy fibrillæ resembling acute myocarditis were found in the heart wall. He does not mention the condition of the coronary arteries.

So close is the relationship between the hypertension and coronary arteriosclerosis that Fahr[15] believes we should consider them together, and that the term "hypertensive heart disease" should connote coronary involvement. In a series of 375 cases of essential hypertension reported by my associates and myself[34] in 1932, it was found that in those cases of long duration the heart weights were greater and the coronary sclerosis was more marked. There was, however, no exact correspondence between the degree of coronary disease, fibrosis, and the cardiac enlargement. Of 171 hearts (group two of that series) forty-four, or 25.7 per cent, had coronary disease of sufficient extent that the thrombosis or pronounced arteriosclerosis was seen macroscopically. As pointed out at that time a more searching examination for sclerosis of the smaller vessels would undoubtedly show much greater incidence.

The relative importance of the high blood pressure and the coronary sclerosis in producing heart disease and failure is difficult to determine. In the past, patients with this type of heart disease came for medical advice after the heart had commenced to show evidence of failure. The blood pressure in these later stages may not reflect the genuine importance of the hypertension, for frequently after the onset of failure the blood pressure falls much below its previous level. It is quite certain from clinical and pathological investigations, however, that very few patients develop a heart failure from the excessive burden of hypertension alone. If pronounced arteriosclerosis develops, heart failure is apt to come at a much earlier date.

Some Phases of Heart Failure

Every patient with persistent hypertension is a candidate for hypertensive heart disease. This does not mean that every hypertensive develops heart failure, but in practically all cases the heart is enlarged. Renal failure, apoplexy, and diseases unrelated to the circulatory system account for the deaths in some of these patients, but sufficient evidence is at hand to state confidently that in more than half of the cases of hypertension, cardiac failure is the main cause of death. Herxheimer and Schulz[25] studied 394 cases of essential hypertension at autopsy and found 384 of them, or 97.5 per cent, had cardiac hypertrophy. Of 420 cases studied by Bell and Clawson,[4] 187, or 44.5 per cent, had heart failure. If they had included the coronary group, 254, or 60.4 per cent, were due to this cause. Fahr[15] states that at least 55 per cent of the patients with high blood pressure die of heart failure. Sixty or 70 per cent of the individuals with so-called essential hypertension are destined to develop myocardial insufficiency and die of heart failure. In an analysis of 375 autopsied cases of essential hypertension, my associates and I[34] found that 188, or 50 per cent, died of heart disease, 10.4 per cent of renal failure, 13.4 per cent of apoplexy, 14.2 per cent of infections and 12 per cent of miscellaneous causes. In another se-

ries of 370 cases studied at autopsy, we have found the following statistics: heart disease 54.32 per cent, renal insufficiency 9.48 per cent, apoplexy 14.05 per cent, infections 11.35 per cent, malignant tumors 6.21 per cent, miscellaneous 4.59 per cent (Table II).

From these and innumerable other reports it may be concluded that cardiac hypertrophy rarely occurs in the absence of chronic valvular disease or chronic pericarditis unless chronic hypertension is present to account for it. It must be borne in mind, however, that when a patient has an enlarged heart without pericardial disease or valvular defect, hypertension, too, may be absent at the time of examination. It is astounding how often it may be shown that patients have had hypertension in the past and yet never were aware of its significance. Frequently they seek medical advice after evidences of coronary occlusion have developed and have practically overshadowed the earlier symptoms of hypertensive heart disease.

The additional burden imposed upon the heart by chronic hypertension usually is carried without any distress for many years. The cardiac dilatation and hypertrophy may be looked upon as compensatory changes and for a variable period of time may be the only evidences that the heart is overburdened; yet, sooner or later, for reasons not wholly understood, the hypertensive heart muscle begins to fatigue and finally fails. As hypertension and arteriosclerosis are changes that tend to develop after the age of forty years, the heart is placed under an unusual strain at a time when, due to natural processes, its strength is declining. Then, too, as the disease progresses there is a tendency for the fixation of the diastolic phase at a higher level, which increases the work of the heart. In addition, the arteriosclerosis which has been advancing hand in hand with the hypertension all these years involves the coronary arteries, which lose their elasticity, resiliency, and their ability to dilate when called upon to do so. Therefore, the muscle fibers of the heart suffer from lack of oxygen and proper nourishment and begin to show signs of degeneration. Thus the hypertensive patient whose heart has become gradually more embarrassed slowly but surely begins to show signs of cardiac fatigue. In the early phases of the disease, when hypertension and cardiac hypertrophy alone are

TABLE II. THE CAUSE OF DEATH AS SHOWN AT AUTOPSY IN 370 CASES OF HYPERTENSIVE HEART DISEASE

Cause of Death	Number	Per Cent of Total
Heart Disease	201	54.32
Apoplexy	52	14.05
Renal insufficiency (uremia)	35	9.48
Infections	42	11.35
Malignant tumors	23	6.21
Miscellaneous	17	4.59
Total	370	100.00

the clinical features, the patient usually fails to realize the true significance of his position. He does not understand that he is on the route which in most cases terminates in heart failure. Occasionally after a short period of overexertion, an episode of dissipation, an emotional upset, or an attack of influenza he suddenly realizes that the heart has been overtaxed. For the first time in the long course of events he becomes aware of an uncomfortable sensation which marks the beginning of the downward trend of the heart's reserve force.

Various explanations have been advanced for the tendency of the hypertrophic heart to fail. Harrison[19] offers an explanation which concerns the rate of oxygen diffusion in the muscle fibers. He considers that the oxygen tension of the arterial blood is insufficient to cause oxygenation of the thickest fibers seen in the thickened heart in the time available during diastole. As he points out, the blood remains in the cardiac capillaries for only a fraction of a second and oxygen diffusion does not occur instantly but requires a definite interval of time. It would appear that a rapid heart rate or too great increase in the distance to which oxygen must diffuse might interfere with complete oxygenation of the central portions of muscle fibers and hence with the recovery process. He further points out that this is why small animals with thin cardiac muscle fibers may have a normal heart rate of three hundred or more and yet not suffer from heart failure.

In recent years there have been attempts to explain the failing heart in hypertension on a chemical basis. Herrmann and his associates[22,23] believe that low myocardial creatine values are more or less constant accompaniments of congestive heart failure and must be among the sig-

TABLE III. THE TYPE OF FAILURE AT THE ONSET OF MYOCARDIAL INSUFFICIENCY IN 269 CASES OF HYPERTENSIVE HEART DISEASE

Type of Heart Disease	Number	Percentage of Total
Right	8	2.97
Left	131	48.70
Total	110	40.89
Undetermined	20	7.44
Total	269	100.00

nificant chemical changes that are associated with myocardial damage and insufficiency. It is pointed out that these low creatine values are probably the result of an insufficient supply of oxygen to the fibers of the heart muscle. After all, no one is able at present to show by study of the heart muscle after death just why it has failed. Recent advances, however, seem to be steps forward in this direction.

The kind of heart failure that occurs in hypertensive heart disease is usually easily recognized. Since the brunt of the added burden is thrown upon the left ventricle, this chamber dilates and hypertrophies. Failure is usually of the left ventricular variety, which is distinguished chiefly by dyspnea, often paroxysmal and usually nocturnal, substernal distress, palpitation of the heart, and cough associated with mild congestion in the bases of the lungs. This type of heart failure is looked upon as the ultimate result of aortic regurgitation and of hypertension. Occasionally so-called right ventricular failure is the earliest manifestation of the failing heart in hypertension. In this form of failure there is edema of the legs, ascites, effusions into the pleural and pericardial sacs, engorgement of the liver, turgescence of the veins of the neck, and cyanosis. Right failure is more apt to be the initial clinical syndrome in cases of mitral stenosis and pulmonary sclerosis. In a later phase, after left ventricular failure has become well established and is resistent to treatment, the right heart may fail too. Total heart failure may then be said to be present. The term congestive heart failure is used to identify either right or left ventricular or total failure.

As Harrison[10] concludes, dilatation and hypertrophy primarily involve only one ventricle of the heart to any marked degree, but ordinarily the other ventricle is affected to some extent even in the beginning. More recently Thompson and White[41] point out that hypertrophy of the right ventricle is often found in persons with pure left sided strain regardless of the presence or absence of left ventricular failure, but if there is failure the degree of hypertrophy is increased. Occasionally left ventricular hypertrophy and failure may be present with no hypertrophy of the right ventricle. In 269 cases of this series of hypertensive heart disease there was an early right ventricular failure in eight cases, left ventricular failure in 131 cases, total failure in 110 cases, and the type of onset was undetermined in twenty cases (Table III).

Most observers are fully aware of the fact that the chief underlying cause of heart failure, the arteriosclerosis and the hypertension, can be influenced only indirectly. Yet more frequently than is usually thought, the actual attack of heart failure is precipitated by some extra-cardial condition. Cough, associated with upper respiratory infection, seems particularly likely to precipitate an attack of failure. Although I am not able to give exact statistics from my own cases, it is certain that such precipitating factors are very common, and, furthermore, that frank heart failure may be retarded or forestalled for some time by the adequate control of extra-cardiac precipitating influences. The first sign of heart failure in hypertension may be a severe attack of acute pulmonary edema, angina pectoris, or even sudden death, but in most cases the onset is insidious and is characterized by the classical features of left ventricular failure.

Overtaxing of the left ventricle in high blood pressure leads to dilatation of that chamber and certain evidences of respiratory embarrassment occur. Orthopnea and paroxysmal attacks of nocturnal dyspnea, often called cardiac asthma, are the early symptoms. These symptoms have been variously explained, but now it is generally agreed that they are the sequelæ of pulmonary congestion resulting from the back pressure effect of left ventricular failure. Such congestion in the lesser circulation may be very mild and difficult to elicit, or it may be so definite that the classical picture of acute pulmonary edema develops with the coughing of frothy pink material, pronounced respiratory embarrassment, and signs of waterlogged lungs. The direct results of these changes in the pulmonary circulation are a diminution in vital capacity of the lung and an

increase in the respiration rate. The various forms of respiratory difficulties may be attributed to the combined effect of these two factors. The discussion of the clinical features of left ventricular failure must take into account two stages of the syndrome, the early one, when the patient is usually seen in the office, and the later one, when he is examined in the hospital or in bed at home. In the earlier stages the patient may be hardly aware that anything is wrong with him. There may be a slight cough associated periodically with blood-tinged sputum. At first the short windedness, which later is such a harassing symptom, occurs only at night or on heavy exertion. Almost subconsciously the patient requires an extra pillow at night for a comfortable rest. Such requirements are at first attributed to nervousness, and only later on does the patient realize his predicament. After a number of weeks or months these vague subjective symptoms become more pronounced, and finally the fully developed clinical picture of left ventricular failure is unmistakable. At this time, periods of dyspnea come more frequently and are of greater duration and severity. Yet even then there may be comparative freedom from respiratory distress for days at a time if the patient remains quiet. Slight exertion, emotional strain, or a heavy meal may precipitate the spells of shortness of wind. Most characteristic are the paroxysmal attacks of dyspnea that come on at night. The patient who has been comfortable during the day goes to bed feeling assured of a night free from distress. After a few hours of sleep he is suddenly taken with a sensation of suffocation and great oppression over the heart. He wakens abruptly and is forced to sit up in bed and gasp for air. Frequently, unless the attack is cut short by an injection of morphine, he leaps from bed to an open window in order to overcome the sensation of strangling. Peculiarly enough, after half or three quarters of an hour of distressed breathing, he is often able to lie back in bed and sleep comfortably for the rest of the night. In the very near future, however, periods of dyspnea, unless retarded with medication, become more and more frequent until finally the patient is never free from the oppressed feeling of suffocation.

Substernal Distress.—As a rule, it is difficult to evaluate the various types of distress in the chest in patients with heart disease. The fairly constant association of the elevated blood pressure and coronary sclerosis of hypertensive heart disease leads to difficulty in assessing the true part played by either of these factors in the failing heart. From a study of the clinical histories, we agree with Levine[31] and Fahr,[15] who believe both should be considered together.

When a hypertensive individual develops cardiac pain of the anginal variety, one may assume that the coronary phase of the disease has begun to take its toll. In our series, chest pain was the initial symptom in thirty-four, or 12.56 per cent, of 269 cases. Coronary arteriosclerosis was present in 158 of 201 cases studied post mortem, or 78.45 per cent. Coronary thrombosis was found in eighteen, or 8.95 per cent, of these cases. Angina was present in 329, or 26 per cent, of 1,249 cases reported by White[46] when they were first seen for treatment.

Providing the physician personally makes an accurate analysis of the pain, true angina may be satisfactorily identified, but if he must rely upon records made by various other examiners, the conclusions drawn must necessarily lack precision. The patient frequently finds it difficult to convey his true sensations to the physician, who may fail to interpret his descriptions properly. Tightness, or pressure in the sternal area, a sensation of choking, or a toothache type of pain are expressions commonly used by patients to describe the sensations. Genuine anginal pain usually is felt in the sternal area, but occasionally it occurs over the heart. Sometimes the distress is mistaken for indigestion because of the formation of gas in the stomach. A person who is over forty-five years of age who develops indigestion and gaseous eructation following meals should be examined for cardiac disease. A pain which is paroxysmal in type produced by exertion, emotional strain or heavy eating, and which tends to disappear under the influence of nitroglycerine is most likely angina pectoris. The significance of this type of pain in these cases is difficult to determine. Occasionally angina may be present for a number of years before other serious signs of heart failure. Although it does not mean that heart failure is present, in hypertensive heart disease the two usually go hand in hand. Clinical observations show that the onset of anginal pain in hypertensive heart disease is

usually followed soon by other evidences of heart failure. If the pain occurs after dyspnea has set in there is usually a rapid decline in the cardiac reserve. It is an ominous sign, regardless of the stage of the disease when it is first seen.

Palpitation, Tachycardia, and Cardiac Irregularities.—Heart consciousness is an early and an important symptom in hypertensive heart disease. Frequently before dyspnea, anginal pain, or other evidences of heart failure, the patient has palpitation. A sense of fluttering or hammering of the heart, which may be either rapid or slow, are the descriptions patients often give. The differentiation of palpitation from actual pain in the heart area is not easy to make. This symptom is more confusing than others because it often occurs in neurotics with perfectly normal hearts; yet I wish to emphasize that the onset of palpitation in a patient with hypertension is a symptom that must be examined carefully. With what frequency palpitation is a feature of the clinical picture of hypertensive heart disease may be estimated from statistical reports. Our own records show palpitation to be the presenting symptom in thirteen of 201 cases, or 4.83 per cent.

Tachycardia often develops in the presence of a hypertrophic heart and usually brings on premature failure. During diastole the left ventricle receives its blood supply and an increase of cardiac rate interferes with the oxygenation of the heart muscle fibers. As stated by Williamson[47] there is no worse associate for a weakened pericardium than tachycardia. Not only does it goad the weak muscle to more frequent contractions, but it deprives the harassed myocardium of adequate nourishment.

The significance and frequency of auricular fibrillation in hypertensive heart disease has recently been studied by Flaxman.[16] In his series, 158, or 25.3 per cent, of 623 patients had auricular fibrillation. In White's[46] group of 1,249 hypertensive cases auricular fibrillation occurred in 170, or 13.6 per cent, of them. In our own series, auricular fibrillation was an early sign of heart disease in thirty-four of 120 cases, or 12.63 per cent. When auricular fibrillation occurs earlier in the course of hypertensive heart disease than other signs of heart failure, it is usually followed by other evidences of congestive failure within a short time. This leads one to believe that Fahr[15] is correct in his opinion that auricular fibrillation may be responsible for the onset of heart failure which otherwise would have been deferred for some time to come. There can be little doubt that the onset of auricular fibrillation renders an already embarrassed heart more ineffective and serves to mark the beginning of cardiac defeat.

Auscultation of the hypertrophic heart often reveals gallop rhythm. The appearance of this triple rhythm, or reduplication of the first sound, in association with cardiac enlargement and hypertension, is an ominous sign which usually indicates that frank heart failure is not far off. It usually occurs at the time when the hypertensive heart commences to show dilatation. A systolic mitral murmur occasionally makes its appearance coincidental with the development of gallop rhythm. With proper management, the gallop rhythm may be controlled for weeks or even months, but it reappears after the heart muscle has become more exhausted. This arrhythmia is one of the surest signs that the overburdened heart muscle is exhausted, and it occurs in the closing period of hypertensive heart disease. Gallop rhythm is never heard in association with auricular fibrillation, but at times it is displaced by it.

Electrocardiographic studies in hypertensive heart disease reveal no clear-cut results. The left ventricle preponderance or left axis deviation is the result of cardiac enlargement of the left ventricle. Bundle branch block indicated by a prolongation of the QRS complex is usually associated with the miliary scarring of the heart muscle. Decided changes in the T waves are usually manifestations of coronary occlusion or coronary infarction. It may be concluded from reports that in certain cases following splanchnicectomy inverted T waves become upright again that these abnormalities may not depend upon such organic changes.

Prognosis

How long a patient with hypertensive heart disease may live before the onset of cardiac insufficiency has not been determined, but as a rule, once the heart has failed, there is little likelihood of its regaining its former reserve force. The general opinion is that hypertension usually exists from ten to fifteen years before the heart

fails, although there is considerable variability. It is difficult to foretell how long the heart muscle will be able to stand the added burden because frequently the time of onset of hypertension cannot be determined. When confronted with a hypertensive patient, it is desirable to determine how long he will live before the hypertrophic heart begins to fail. Without an attempt at accuracy there are several measures to be used in approximating this information. As a rule, a diastolic pressure of 120 to 140 connotes a worse outlook than the lower ones (90 to 120). The greater the cardiac enlargement, the sooner heart failure sets in. Progressive enlargement of the heart is a poor prognostic omen. The degree of arteriosclerotic change seen with the ophthalmoscope is helpful in prognosis. Retinopathy with edema of the discs is a grave sign, but without choked discs it is not so serious. Electrocardiographic examinations may show the presence of bundle branch block or inversions of of the T waves in Leads one and two or prolongation of the Q wave in Lead three. These abnormal configurations are not incompatible with longevity and may be present for years before cardiac failure develops. In the hypertensive hypertrophic heart, as in other types of heart disease, there is no accurate method of determining the amount of reserve force the heart muscle has. Before certain signs and symptoms indicate that cardiac strength is failing, we do not know how long the heart muscle can withstand the extra load of hypertension. The kind of work the patient does, the type of treatment he receives, as well as the intrinsic qualities of the heart muscle, all play a part in prognosis.

The duration of life following the onset of cardiac symptoms is a subject of considerable interest and also of great variation. In a clinical analysis of our cases it was found, as shown in Table IV, that ninety-six, or 35.6 per cent, of the total number of patients lived from one to five years after the first signs of heart failure set in. Ninety, or 33.4 per cent, of them lived less than six months. Twenty-seven, or ten per cent, lived from five to ten years. Only two, or 0.74 per cent, lived ten years or longer after the first sign of failure. The unusually high mortality rate in the first year following the onset of cardiac symptoms may in part be explained by the fact that the patients were of the type seen in charity institutions. Perhaps in private practice, with more careful regulation of the patient's routine, the mortality rate would be much lower. Notwithstanding the circumstances under which the patient is treated, it is undoubtedly true that if anything is to be done in the way of prolonging the life of individuals with hypertensive heart disease, vigorous therapeutic measures must be established before the onset of cardiac breakdown. In consideration of the prognosis, it is important to remember that the hypertensive cardiac is subject to malignancies, acute abdomen, and all the diseases that tend to occur in the individual without hypertensive heart disease.

TABLE IV. DURATION OF LIFE FOLLOWING ONSET OF CARDIAC SYMPTOMS IN 269 CASES OF HYPERTENSIVE HEART DISEASE

	Number	Percentage of Total
Less than 6 months	90	33.45
6 mo. to 1 yr.	27	10.03
1 yr. to 5 yrs.	96	35.68
5 yrs. to 10 yrs.	19	7.06
10 yrs. or over	2	0.74
Time of onset unknown	35	13.04
Total	269	100.00

Finally, clinical observations show that in hypertensive heart disease, as in chronic nephritis, the clinical course is not featured by a steady decline of reserve force. There are periods of remission alternating with relapses which last from a few weeks to several months. During a remission the patient's hopes are buoyed up, and during a relapse he becomes unduly despondent. If these features of the course of the disease are understood by the physician in charge, the prognosis will be made more guardedly and more satisfactorily than otherwise.

Treatment

Every hypertensive must be considered a potential case of heart failure. In hypertensive heart disease there is a lapse of a period of years between the beginning of cardiac enlargement and frank failure. In treatment, then, one must consider the appropriate therapeutic measures to be used in the stage preliminary to frank failure and those to be used after the heart has begun to fail. The hypertensive patient usually feels too well to resort to an exacting medical routine in the early stages of the disease, and frequently

even in the transitional period which occurs between marked cardiac hypertrophy and failure. It is most important in the early stages of the disease to guard the patient's heart against those influences which tend to precipitate genuine heart insufficiency. In spite of the fact that the patient feels comfortable, the physician must bear in mind that the hypertensive with cardiac enlargement is a candidate for heart failure and that in 50 to 60 per cent of the cases this disorder will terminate his life.

Too often the patient in the early stages of hypertensive heart disease is allowed to drift on without taking advantage of measures that may retard the progress of the disease. Frequently, too, the disease has advanced almost to the closing period of its course before the patient seeks medical relief. Vigorous therapeutic measures at this time can only be palliative, and as a rule are incapable of stemming the downward trend of the disease. Periodic health examinations, which have been advocated, are of great help in detecting hypertension in its early stages, and therefore should help prevent the heart failure of later years. Yet this ideal of a periodic examination, with the object of preventing or retarding the degenerative diseases, has not been realized, for too few people take advantage of this type of an examination. It is questionable whether lecturing to the public or campaigning among the laity for more health examinations would be as effective in controlling this disease as placing more emphasis upon it in teaching students, internes, and house physicians. Enlightenment must emerge from a better understanding of the disease by the physician. After he has been fully convinced of the importance of retarding and forestalling the remote sequelæ of this disorder, he will put into practice effectively what is aimed at in all preventive medicine campaigns. It is no exaggeration to say that thousands of patients with hypertensive heart failure are at the close of their life's career and almost at the end of life itself because the importance of hypertension and its effects upon the heart were not appreciated early enough. If we are able to visualize heart failure as an end-result in all the hypertensives that come under our influence, and if, therefore, we treat them accordingly, morbidity and mortality from heart failure is sure to decline.

In the past there were, and there still are, prominent clinicians who fail to stress the importance of a benign essential hypertension upon the patient's mind for fear of making him neurotic. One may well neglect the unfavorable effect that is brought about by a few patients becoming heart conscious, if many others are saved from cardiac breakdown. In brief, then, every hypertensive is a potential case of cardiac failure, and physicians into whose hands these patients come should be alert to the remote consequences of the disease and should develop that skillfulness and ingenuity which are necessary to impress the patient who is sick and does not realize it with the importance of frequent examinations and the requisites of treatment.

General measures such as rest, relaxation, and more adequate recreation are therapeutic aids often not taken seriously enough. Obesity and overweight should be combated vigorously, for these aggravate an already overburdened myocardium. A diet which keeps the weight under control is adequate for the treatment of the hypertension too. Sedatives are often used and are effective in the control of the hypertension in the early stages.

The successful management of the patient requires more than therapeutics aimed at the hypertension and the heart. In no type of heart disease can the heart be singled out from the remainder of the body and treated alone. The influence of food intake, gastro-intestinal sluggishness, psychic changes, overweight, insomnia, and numerous extra-cardial conditions is of outstanding importance in the course of the disease.

In the early stages, before frank myocardial insufficiency has occurred, specific treatment must be considered. As stated by Christian,[8] cardiac enlargement is functionally harmful, and intelligent therapeutics will retard it. He recommends the use of digitalis to delay the appearance of heart insufficiency. Doses of 1½ grains twice a day are effective unless some contraindication occurs. This digitalis therapy is continued throughout the life of the patient. Before outspoken evidences of failure, the effect upon the hypertrophic heart of such conditions as upper respiratory infection, overexertion overeating, mental anxiety in precipitating heart failure must be emphasized. It is surprising to find what a large number of patients date the onset

of failure to one of these extra-cardial factors. Coughing, with upper respiratory infections, throws an added burden upon the heart, and this should be controlled whenever possible.

The treatment, after the classical textbook picture of cardiac insufficiency has developed, usually is administered with more rigor and care than that given in the earlier phases of the disease. In this later stage it is difficult for the heart muscle to regain its lost reserve force. Under treatment, however, certain of these patients respond and appear to make complete recovery, but within six weeks to six months the same symptoms of failure reappear. It is desirable to do all in our power to bring about a remission in the course of the disorder. If this attempt is successful, the patient may be symptom-free for a number of months or even years, depending on how carefully therapeutic measures are followed. There is, however, no accurate way of measuring the ability of a heart muscle to regain its lost reserve force.

Therapeutic aids in this stage may be summarized as follows: (1) prolonged rest in bed; (2) digitalis; (3) diuretics; (4) oxygen; (5) sedatives. Regarding rest, there is no particular rule except that the patient must be kept absolutely in bed for a period of weeks, and preferably months, for clinical experience has shown that prolonged rest is the most important measure in regaining the cardiac reserve. Digitalis is indicated whether arrhythmia is present or not. Even though there is bradycardia, digitalis should be given when the heart is failing. Doses ranging from 3 to 7½ grains a day are indicated. The exact amount of digitalis necessary and the methods of administration are problems that must be decided in each individual case. Sometimes patients tolerate the tablet better than the tincture or capsule, and vice versa. Nausea and vomiting are not always produced by the digitalis, which frequently is blamed for it.

Diuretin, novasurol, and salyrgen are the chief diuretics in use. More recently diuretics such as salyrgan and mercuropurin have been made in suppositories, and this method of administration is often very effective and desirable. A point of practical importance that is doubtless well known, but, I believe, frequently overlooked in the advantageous use of diuretics, is that peripheral edema is not the only indication for the administration of a diuretic. Frequently this form of therapy makes the patient with no palpable edema, but with dyspnea, pulmonary congestion and cough, more comfortable. Hope,[26] over a hundred years ago, had this to say regarding the use of diuretics in the treatment of hypertrophy of the heart: "In addition to purgatives, I have seen the most decided advantages result from diuretics, and not only when there was dropsy but also when there was none." One must remember, too, that albuminuria is not a contraindication for the use of mercurial diuretics in these cases, for their administration is often followed by a disappearance rather than aggravation of this condition.

One of the modern additions to the treatment of heart failure is the widespread use of continuous oxygen inhalations. Patients are made much more comfortable and periods of remission are seen to occur sooner when oxygen is commenced early and continued until the patient is free from the harassing symptoms of dyspnea, orthopnea, and insomnia. Of the three methods of administration, the oxygen chamber, the oxygen tent, and the nasal catheter, the first is undoubtedly the most effective, but the last is the most practical. The oxygen tank with humidifier and nasal catheter can be used whether the patient is at home or in the hospital. Experience with this method of giving oxygen proves that, although it has its drawbacks, it has many advantages and will perhaps be the best method of administering oxygen.

Concerning sedatives, it is important to emphasize that a minimal amount of drug therapy is desirable. Yet at times the dyspneic patient requires a large dose of the most effective of all sedatives, morphine sulfate. Because of its untoward action upon the gastro-intestinal tract and other organs, it should be reduced in amount and discontinued at the ealiest possible moment. I have never thought that the danger of morphine addiction is a serious matter in these patients. A substitute for morphine should be introduced after several days of treatment. Such preparations as codein sulfate, amytal, chloral hydrate, and bromides are frequently effective. The sedation is important because it controls insomnia, relieves coughing, and brings about physical and psychic rest to the anxious and fatigued patient.

Summary

1. Hypertensive heart disease occupies a unique position in the classification of heart disorders, for it is not only the most common type of heart disease, but is one of the least understood. Its true significance has only recently been recognized by a few leaders in this field of work. This disorder is not only the most common type of heart disease, but it occurs more frequently than all other forms put together. A review of vital statistics does not properly reflect its importance. Considerable confusion in this regard is undoubtedly due to the lack of appreciation of proper terminology. Obsolete, inaccurate, and vague terms used to designate this type of heart disease have been a stumbling block in its more widespread recognition.

2. Hypertensive heart disease is the result of the combined action of hypertension and arteriosclerosis of the coronary arteries. The heart which has been overstrained by persistent hypertension hypertrophies, becomes impoverished by lack of proper nutrition due to coronary sclerosis, and fails as a result of a combination of these two forces.

3. Hypertensive heart disease is not a disease in the same sense as typhoid and tuberculosis. It is the result of two other conditions, hypertension and arteriosclerosis, and the cause of them is not known. It is almost traditional to hold our modern mode of living responsible for some of these degenerative diseases, yet it does not seem that we work any harder or longer or dissipate to any greater extent than our ancestors. There is proof, too, that these conditions existed in ancient civilizations. It is agreed, however, that there are two prominent factors in the etiology of hypertension and arteriosclerosis, namely hereditary disposition and advancing age.

4. The hopes of those physicians of fifteen years or so ago that periodic health examinations would lead to earlier diagnosis and treatment of these progressive degenerative diseases, have not been realized. Such examinations would be ideal, but too few people take advantage of them. Emphasis on these conditions must emerge from a better understanding of them by physicians themselves. In medical schools and in the hospitals the future practitioners should learn what the remote sequelæ of a benign hypertension are. Finally, the physician in practice should be encouraged to develop the skillfulness and ingenuity necessary to keep a patient with a mild hypertension, who is neither sick in his own mind nor well in the doctor's, under constant medical supervision for years in an effort to forestall the remote consequences. In this way the progress of hypertensive heart disease will be retarded and the morbidity and mortality rates may be reduced. The fuller realization that every hypertensive is a subject for ultimate heart failure is the key to better safeguarding the heart's health.

5. Of 269 cases studied clinically and at autopsy some degree of coronary sclerosis and myocardial fibrosis was present in 78.45 per cent. The cause of death as determined by autopsy was heart disease in 54.32 per cent of the cases, apoplexy in 14.05 per cent, renal insufficiency in 9.48 per cent, intercurrent infection in 11.35 per cent, malignant tumors in 6.21 per cent, and miscellaneous in 4.59 per cent.

6. In this study it was found that 80 per cent of the patients died within five years of the time of onset. This emphasizes the futility of postponing treatment until frank failure sets in. Every hypertensive must be looked upon as a candidate for ultimate failure, which as a rule occurs within five to fifteen years after the onset of hypertension. In the transitional stage, that is the period that elapses between hypertrophy of the heart and heart failure, treatment is most effective.

7. Statistical analysis is made of the chief clinical features and the frequency of their occurrence is discussed.

8. The treatment of hypertensive heart disease is considered according to its two main periods: (a) before the evidence of frank failure sets in, and (b) after myocardial insufficiency occurs. During the earlier period, which may vary from months to years, the main object of treatment is to safeguard the hypertrophic heart from influences which precipitate heart failure and to retard the progress of the disease as effectively as possible. To accomplish this it is desirable to avoid infections, especially of the upper respiratory type, overeating, overweight, overexertion, overwork, and to encourage prolonged periods of relaxation and rest. Digitalis and sedatives judiciously used in conjunction with the other therapeutic measures may be

effective in postponing the final outcome for many years. The treatment of the case after the heart has failed consists of the use of digitalis, diuretics, oxygen, and the other methods usually employed in any case of heart failure.

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HEALTH PROBLEMS FROM THE LAYMAN'S POINT OF VIEW*

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Why should a layman discuss solutions to health service problems? First, because the layman receives all the health services; second, because he pays for them. The first fact makes him very much interested in quality. The second makes him very much interested in cost. The layman's specialty in the medical care field is that of payment.

Why do people talk so much about sickness? What are the economic factors which make health service so much different from transactions involving other commodities such as shoes, ships, sealing wax, automobiles, radios, or tobacco?

The Economic Aspects of Health Service

Health services have been regarded from time immemorial as something a person should receive, whether he could pay for them or not. This fact is taken for granted by the general public and the medical profession. In early days when only one kind of medical practitioner was in the field, the public assigned the responsibility for health service to the doctor in some such fashion: "Doctor, you take care of the people and we will take care of you. If you find someone who cannot pay, serve him according to his needs. If you treat someone who can pay you well, collect from him according to his ability."

As a result of this practice, there has developed throughout the medical care field a so-called sliding scale of physicians' fees. The average practitioner has, however, enjoyed merely the privilege of rendering free work and sliding the scale downward. Only rarely, if ever, has he enjoyed the privilege of assessing and collecting high fees from people of ample means.

Health services are compulsory. One does not buy miscellaneous amounts of health services in the way he would buy a theatre ticket, a bunch of roses or even a bottle of patent medicine. To be sure, there are some people who enjoy ill health. But they cannot be regarded as typical of the average American citizen. Most of us want our medical care for hygienic rather than for esthetic purposes.

Sickness and its cost are unpredictable as to time and amount. No one can tell when he will be sick or what his sickness will cost him. If we knew when we would be sick, we might do something about it, like the man who expressed a wish to know where he was going to die. A friend asked, "Why such morbid curiosity?" The man replied, "I would never go near the place."

If we knew exactly when we were to be ill and what the illness would cost us, we would plan in advance. But sickness does not follow the calendar or respect the pocketbook. It does not wait until the baby is paid for, before bringing a fractured limb to some other member of the family.

Health service is mysterious, an economic commodity of which the buyer is absolutely no judge of quality or need. A patient does not really know either before or after receiving medical care, whether he has been properly served. This responsibility and knowledge rests entirely with the practitioner. Do not misunderstand me. This arrangement is necessary and desirable, in my opinion, and it will be a sorry day when the patient starts diagnosing and treating his own disease. Even a physician does not attempt this absurd procedure. A significant fact, however, is that medical care, being mysterious in nature, represents a type of transaction in which the buyer or the patient is entirely at the mercy of the seller or practitioner. This condition requires a very high standard of ethical policy or procedure, and it is fortunate for us, as laymen, that the ideals of the medical profession have been maintained at such satisfactory levels in most instances, for the physician not only decides whether or not we should receive care, but he has the privilege, unique in the social order, of determining how much the buyer should pay.

Health service is intensely personal. No one knows this better than the patient. It is the only

*Read at the annual meeting of the Minnesota State Medical Association, St. Paul, Minnesota, May 4, 1937.

type of service for which the patient cannot use a substitute. A busy executive cannot send his assistant manager to the hospital in his place. A solicitous parent cannot take over the sufferings of his child with an infectious disease. There is no substitution in the field of medical care.

The personal nature of health service has two important economic influences. First, the patient feels that no standard of quality can possibly be too high. The best is none too good. This explains why patients are willing to pay large sums for treatment by certain physicians, even though scientific judgment might suggest that adequate care could be obtained from other persons. The second economic influence is the desire of the patient to choose his own physician. Free choice of doctor, particularly for specialized services, is probably of more advantage emotionally than scientifically. Undoubtedly one's family doctor could choose a surgeon more appropriately than a patient could do for himself.

Medical care has no exchange value. Health services cannot be transferred to someone else or returned to the original vendor. A successful surgical operation may restore working capacity which will bring an income of thousands of dollars, yet the former patient cannot exhibit his scar as collateral for a loan at the bank.

The receipt of health services interferes in the short run with the ability to pay for them. Absence from gainful employment coupled with other types of expenses in the home at the time of sickness make health services particularly difficult to purchase.

I have cited the various characteristics which explain why the layman feels the way he does about medical care, namely, that health services are essentially social services, that they are compulsory, are unpredictable as to time and amount, are mysterious, are personal, are of no exchange value, and tend to interfere with the patient's ability to pay for them.

The patient specializes in paying for medical care. This is essentially and uniquely his field, and he should not presume to judge the scientific value of health service, which is a task of the medical practitioner, particularly the physician. The laymen's task is to pay the costs of health service and to pay them under such conditions that the medical profession and other professions can render services of high quality. Conversely, the primary responsibility of the healing profession is to render good health service and to insist on methods of payment which will not interfere with standards of quality. The layman specializes in payment; the medical profession and the allied groups specialize in the provision of health service.

The Cost of Medical Care

The health problem from the layman's point of view is not to reduce the costs of medical care, but to meet them. Probably the total expenditures for health services should be increased. Approximately 4 per cent of the national income is spent for the prevention and cure of disease, or if we wish to use the metaphor, for the maintenance of the population. Certainly 4 per cent of the national income to insure good health, the very basis of good life, cannot be regarded as too large a total or proportion for this purpose.

Much of the money now being spent for health services is wasted. The expenditures for patent medicines and for non-medical healers alone would suffice to operate all of the general hospitals in the United States, including those conducted by the federal government, state, city and county, as well as the voluntary associations and private hospitals. More than a half billion dollars annually is spent for patent medicines and various types of non-medical healers, much of which, if not actually wasted, certainly is of very little economic or professional value.

If people were as careful about paying medical and hospital bills as paying for radios, automobiles and tobacco, there would be no complaints about the cost of medical care. But "being careful" implies that it is possible to be careful. This requires a special type of budgeting for health services which does not apply to ordinary commodities.

It is impossible for an individual to budget for his medical care, even to plan soundly for the expenses of a childbirth in the family. Suppose, for example, that monthly installments of $10 are paid into a hospital for the cost of a maternity case. There is nothing to prevent a new expenditure for some other hospitalized illness consuming the entire amount accumulated for the maternity case. Sickness is not like other economic experiences.

Imagine the following fantastic situation. You are in your living room playing the radio. A rap on the door reveals a radio dealer with a new instrument under his arm. The following conversation takes place:

Radio Dealer: "Here is a new radio."

You: "But why do you bring it to me? Is it a present from Uncle Henry?"

Radio Dealer: "No, no . . . I am selling it to you. The price is $70.20.

You: "Oh, in that case, I don't wish to purchase it."

Radio Dealer: "But you must."

You: "I can't afford it."

Radio Dealer: "I will leave it here and expect you to pay for it immediately or by installments."

The experience just described would not occur in the radio field, but it happens in the field of medical care continuously. The costs are compulsory and unpredictable as to time and amount.

Hospitalized illnesses are particularly difficult to pay for. Not only are they large in total amount, but they are also accompanied by absence from gainful employment. Hospital bills are not too large, considering the value received. One receives more for his money in the average American hospital than in a hotel, but nevertheless, an amount of $50 or $150 is difficult to pay under any condition.

How then are people to place hospital care and other expensive illnesses in the family budget if they cannot predict either the amount or the time? The answer is that some procedure must be used to remove the uncertainty and economic hazard of hospitalized illnesses. This can be done by having each individual pay an average amount into a common fund to apply on his own hospital bill, if he requires such care, or upon someone else's hospital bill, who requires it. Approximately 10 per cent of the public require some hospital care each year. The uncertainty can be removed by having everyone pay an equal amount into a common fund for the bills of those who require such care.

Group Hospitalization in the United States

Group budgeting of hospital bills is not merely an idea. It is an accomplished fact at the present time with more than a million subscribers paying amounts ranging from 50 cents to $1 per month per individual into a common fund for the payment of their hospital bills.

The Minnesota Hospital Service Association is the second largest plan in the United States with more than 100,000 employed persons and their dependents eligible for hospital service benefits.

It is my responsibility to serve as adviser and counsellor to newly-formed plans in the United States and to attempt to ensure that they are established along lines that are professionally and economically sound. On May 1 there were 350,000 subscribers to the New York City plan, 55,000 in Cleveland, 65,000 in Rochester, New York, and others with smaller enrollment in plans more recently formed.

The American Hospital Association is an association of hospitals. The Association has responded to the general demand by the public for group budgeting of hospital bills by attempting to study and guide the movement. Certain characteristics or criteria have been established which should be present in any plan wishing to serve the community to best advantage. These criteria may be mentioned briefly at this point. They were established in February, 1933.

First, primary emphasis should be placed upon the public welfare rather than upon hospital finance. Group hospitalization is a way by which people pay hospital bills, not a way by which hospitals pay their bills.

Second, the plan should be organized and sponsored on a non-profit basis. No private organizer or promoter should risk his own funds in a project of this type. It should be owned and sponsored by the community and the hospitals which provide this service.

Third, the benefits should be limited to the services provided by the hospitals themselves and not include physicians' or surgeons' services.

Fourth, there should be free choice of hospital by the subscriber at the time of care, and the contract should not interfere with the subscriber's choice of hospital, his relation to his physician, or the physician's relationship to the hospital.

Fifth, the plan should be economically and actuarially sound. Rates should be high enough to provide the services required and to remunerate hospitals adequately for services required.

Sixth, promotion and administration should be

on a dignified basis with the emphasis on group budgeting rather than upon the merits of the participating hospitals.

Group budgeting is, in the final analysis, a form of insurance and is regulated definitely by insurance laws in New York, Illinois, California, Massachusetts, Maryland, Alabama, and Pennsylvania. A number of states such as Minnesota and New Jersey regulate these plans under laws other than those administered by the department of insurance. In every instance, they should receive the benefit of regulation by representatives of the community.

Where plans have been established for the community welfare, they have been successful. No non-profit, free-choice hospital service association has been discontinued, although a number of single hospital plans and those operated for private profit have been unsuccessful to the promoters as well as to the subscribers and the hospitals.

Hospital insurance plans have definitely aided in permitting patients to receive private hospital care whereas, otherwise, they might be forced to use a public tax-supported institution.

Group hospitalization is not a panacea either for the hospital or the subscriber. It is merely one way of budgeting a part of the cost of hospital care. Group hospitalization will not expand to include physicians' services unless the request and demand for such procedure comes directly from the medical profession. There is very little evidence of any such desire at the present time, and neither the hospitals nor the American Hospital Association wishes or intends to sponsor such a development at the present time.

The public owns the hospitals and the public uses the hospitals. If the hospitals are to be adequately supported and the medical practitioners are to be adequately maintained, the public must be prepared to pay more liberally for a good quality of health service. The average incomes of physicians, dentists, and nurses are not, in my opinion, sufficiently high on the average to remunerate these medical practitioners for their services.

It would seem that more adequate payment for health services can be developed through the use of the principle of insurance by the employed population. For the indigent and unemployed, the use of philanthropy and taxation will have to be continued. Even the money we already spend and will spend in the future could be distributed more wisely to medical practitioners and institutions with resultant savings in health and money through the elimination of the purchase of services from quacks and unqualified practitioners.

Prediction for the Future

The following statements are my opinions as to the way in which the economic aspects of health service will develop during the coming years.

In the first place, I believe that the voluntary budgeting of hospital bills will grow rapidly to include a substantial proportion of the employed population. Low cost hospital insurance plans for people with low and irregular incomes will need to be developed for industrial workers and for rural areas.

Second, it is my opinion that there will be a gradual development of the payment of private practitioners for the care of the indigent, not only in the tax-supported public hospitals, but also in the clinics and wards of voluntary hospitals.

Third, it would seem reasonable to expect a continuation of the present tendency to use tax funds for the care of certain types of expensive illnesses, such as venereal disease, crippled children, cancer and disabling illnesses which involve long periods of treatment and involve heavy expense. There will probably be no reversal in the present trend of caring for nervous and mental and tuberculosis cases from the general tax budget.

The development of health insurance for physicians' services does not appear imminent as far as a national program is concerned, for the following reason. If the physicians are paid for the care of the indigent by the use of government funds, it will remove one of the pressures upon them in establishing charges for pay patients. As far as the general practitioner is concerned, health insurance would be of only limited value, for many patients could pay the small amounts of $2, $3 or $5 per visit. The real difficulties are with the expensive illnesses where charges are from $50 to $200. Even this difficulty will be lessened if hospital bills are placed on an in-

surance program and it the care of the indigent is handled through taxation. The medical specialist will then be able to use the sliding scale in a much more intelligent and satisfactory manner.

Conclusion

The layman's responsibility is that of paying for medical care, not of advising professional groups in the practice of medicine or their attempts to maintain quality. The people want a good quality of medical service. They are not frightened by the use of the phrase, "state medicine," but they are dreadfully afraid of the phrase, "bad medicine." The primary interest of the professional groups is to concentrate on methods of rendering good service, leaving the methods of payment primarily to the lay public, provided such methods do not interfere with the prerogatives which enable a man to practice a high quality of medicine and to conduct himself according to the best professional standards.

VISUAL IMPAIRMENT DUE TO NEGLECT*

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IN THE time allotted, I shall concentrate upon only a few of the outstanding causes of visual impairment which are preventable.

During the past year at the University of Minnesota we have undertaken a census of the blind of this State. Data have been accumulated from the State Board of Control register for the blind, from records of the State School for the Blind, from the register of the Sight Saving classes in St. Paul, Minneapolis and Duluth, and from other available public and private sources. The work is still incomplete. Sufficient material has been gathered to demonstrate the value of preventive measures. Table I, kindly furnished me by Dr. Charles E. Stanford of the University Staff, shows roughly the etiological grouping. Note that hereditary and inherited anomalies constitute practically one-fourth of all actual blindness.

TABLE I. CLASSIFICATION OF CAUSES OF BLINDNESS

	Causes	Per Cent
Congenital and hereditary defects	420	24.10
Infectious diseases (local and general)	319	18.30
Injuries	235	12.80
Glaucoma	159	9.10
Cataracts	157	9.00
Noninfectious systemic diseases	56	3.20
Miscellaneous	265	17.10
Unclassified	112	6.40
Total	1723	100.00

In the juvenile group admitted to the blind school the percentage of blindness from this cause is even higher. This is shown in Table II, which is compiled from statistics furnished by Mr. J. C. Lysen, Superintendent of the Minnesota State School for the Visually Defective.

TABLE II. ADMISSION TO MINNESOTA SCHOOL FOR THE BLIND, 1926-1936 INCLUSIVE

	Causes	Per Cent
1. Congenital and hereditary, including cataract, congenital glaucoma, albinism, aniridia, colobomata, ectopia lentis, retinitis pigmentosa, defective globes and retinochoroidal pathology	136	38.53
2. Optic atrophy from syphilis, hydrocephalus and various undetermined causes	39	11.05
3. Interstitial keratitis and other luetic causes	38	10.76
4. Injuries	37	10.48
5. Ophthalmia neonatorum	29	8.22
6. Trachoma	2	.56
7. Refractive errors	31	8.78
8. Infectious diseases (measles, etc.)	24	6.80
9. Other causes—unclassified	17	4.82
Total	353	100.00

Each of these tables illustrates the importance of consideration of this first and largest group. Under my personal observation have come many family groups with inherited visual defects. To cite a few specific examples: four families with two or more children blind from congenital glaucoma; six families in which congenital cataracts are common through at least three generations, one family in which four children have gyrate

*From the Department of Ophthalmology, Medical School, University of Minnesota. Read before the annual meeting of the Minnesota State Medical Association, St. Paul, Minnesota, May 3, 1937.

choroiditis with high myopia and defective vision; one with two cases of aniridia; another with three children showing colobomata of the choroid and iris; four families with several members afflicted with albininsm; and innumerable family groups, with some offspring developing pigmentary retinal degeneration all of whom will eventually become blind. One family has four children totally or partially blind who have developed retinal detachment following congenital high myopia. I recently reported several family groups with dislocated lenses associated with growth anomalies.[3] Every ophthalmologist could record similar observations. In 1,204 families in which hereditary blindness prevailed, Loeb found that 60 per cent of the 4,155 children produced had inherited eye defects.[2] Park Lewis found 2.5 per cent admissions to the New York School for the Blind were offspring of parents both of whom were congenitally blind.

As Berens[1] stated, "Truly hereditary blindness is that which is transmitted through the germ plasm from parent to child.—It is transmissible in the same way that the color of the eyes and the state of the features are transmissible—It is safe to say that parents who are themselves blind from hereditary causes, or who have in either family cases of hereditary blindness, should not risk having children."

There should be more advice and public discussion relative to prevention of blindness through birth control. Sterilization of mothers who have borne two children with extreme eye defects is entirely logical. The comparative hazard of surgical sterilization is no greater than that of parturition. We as physicians have a duty to perform in giving timely advice in the matter of marriage and birth control to parents of blind children and to persons who are themselves blind. The blind themselves should be warned of the danger to their children in case of marriage.

Transmitted syphilis is the cause of approximately 15 per cent of admissions to the blind school. This figure may be eventually reduced by means of public education, general use of free serologic tests, and when improved therapy with heavy metals over a prolonged period is fully utilized. The nation-wide public health activity to eradicate syphilis by making it a purely medical instead of a social problem should and doubtless will reduce the high percentage of visual impairment from optic atrophy, interstitial keratitis and uveitis due to transmitted syphilis. Routine serologic tests during early stage of pregnancy, when treatment is of value to both mother and child, would prevent many of the ophthalmic tragedies quite as effectively as routine Crede treatments has aided in eliminating ophthalmia neonatorum. Incidentally, blind school admissions due to ophthalmia neonatorum have dropped from 30 per cent to 8 per cent in twenty years. The curves of admission from syphilitic causes and ophthalmia neonatorum in our Minnesota School for the Blind have been widely divergent; the former rising almost 200 per cent, the latter gradually decreasing over a period of ten years. At present there is only one pupil in the Minnesota School for the Blind admitted because of ophthalmia neonatorum.

Volumes could be written upon the subject of ocular manifestations of acquired syphilis. The optic atrophy of tabes and general paresis, together with the frequent ocular palsies accompanying these and other manifestations of cerebro-spinal syphilis, are well known. Even when these conditions are well established, treatment is by no means useless.

Iritis, uveitis and retinochoroiditis due to lues are so frequent in ophthalmic practice they only emphasize the need of routine serologic tests, widespread education of a public ignorant of the necessity not only of prophylaxis but also of persistent therapeutic measures in this disease and the responsibility of our profession in making syphilis a reportable disease. When one considers what has been accomplished in the prevention and control of tuberculosis, the outlook for syphilis and for the prevention of blindness due to this cause is by no means discouraging.

The largest cause of blindness in this country, if monocular injuries are included, is in connection with industry and sports. Large corporations and employers have long recognized the tremendous financial loss resulting from compensation for eye injuries, which constitute 10 per cent of all industrial accidents. In a survey of 583 plants, covered by a study made by the National Society for the Prevention of Blindness and the National Safety Council, it was estimated that the saving through protective goggles amounted to a total of over eighteen million dollars with an additional saving of ten million dollars in wages to some 7,431 injured employes

who suffered shattered lenses, with saving of the eyes. The fact that the General Electric Company, in a ten year period with a constantly increasing force of employes, was able to reduce the number of serious eye accidents from 240 to 7 through the compulsory use of goggles in hazardous occupations is significant and sufficient evidence of their value.

It is not the large corporations and employers who need to be educated as to the value of preventive measures. It is mostly in small industries, in agriculture, and in sports, that we see the largest percentage of damage done through neglect of proper precautions. Further education of the public regarding the value of protective goggles and the consequences of neglect of using them will reduce the high incidence of ocular injuries.

The advantage of non-shatterable lenses should be more widely proclaimed. Every one-eyed person should have a non-shatterable lens in front of his good eye or even plain glass if no refractive correction is needed. Several instances have occurred in my experience where individuals have suffered the loss of or damage to their better eye through foreign particles entering the globe or from ulceration following corneal abrasions or wounds, which could have been averted by the use of non-shatterable lenses. Wearing of well-fitted goggles, with either the extra-tough glass now being developed by certain optical companies, or with non-shatterable glass, affords protection which is entirely worthwhile and should be compulsory for certain eye hazards.

All visual impairment is not due to accident. The removal of eye strain and fatigue are factors of equal importance. Competent correction of substandard vision and eyestrain by lenses should be more frequently required of employes. Even when individuals themselves are well informed as to the value of this procedure, they are not usually as well informed regarding the merits of different types of examiners. Lenses which are ill-fitted or prescribed after an improper measurement of a refractive error produce quite as much fatigue and ultimate visual impairment as no lenses at all. Every ophthalmologist can offer abundant testimony to the fact that fundus lesions from systemic or ordinary ocular disease are rarely discovered in examinations by inexpert hands.

The question of efficient illumination is one which looms large, not only in industrial work, but in home lighting. Ophthalmologists probably err frequently in considering only the refraction errors, without inquiring into the type of work, the quality of lighting, or the elimination of glare, in connection with eye discomfort for which patients consult them. Definite standards of illumination are becoming established through the joint work of the Illuminating Engineering Society, and the American Institute of Architecture. Light meters will gradually become a universal means of determining adequate illumination.

Glaucoma is the attributed cause of one-seventh of adult blindness among the 65,000 afflicted persons recorded in our last census. In Minnesota the incidence of glaucoma among applicants for pension under the Social Security laws is approximately 12 per cent. How may this figure be broken down?

It is true that diagnosis of chronic glaucoma simplex is a somewhat technical procedure. Failure to recognize it at a proper stage for effective treatment, or before one eye has become blind, is due to several causes. One of these is the resignation to decrescent changes by many individuals past middle life who simply accept visual failure as one of the penalties of age. An improper diagnosis of cataracts in occasional instances with advice to delay treatment, or failure to seek expert attention until vision is quite lost, is the reason for lack of success in treatment. Very frequently ignorance and a false idea of economy, engendered by newspaper or show window advertisement of "Eyes examined free," lure many individuals into examinations where salesmanship far exceeds competency. Glaucoma fails of recognition very largely because medical men neglect and optometrists and opticians do not possess the ability to make ophthalmoscopic examinations, to test the intraocular tension and check the fields of vision, often because central vision remains normal. This leads to tragic consequences. Glaucoma can be detected *only* by a thorough examination of the optic nerve, testing the tension of the eyeball and testing the visual fields.

Not infrequently serious damage results also because ophthalmologists fail to impress upon patients in whom incipient glaucoma *has been*

properly diagnosed, the serious consequences of neglect, and the great necessity for periodic examinations and persistent treatment. Frequently the patient becomes careless until *he* vision. He has paid the penalty of a "free examination."

Case 2.—John S., aged sixty-two, began to lose vision in the right eye at the age of fifty. He consulted an

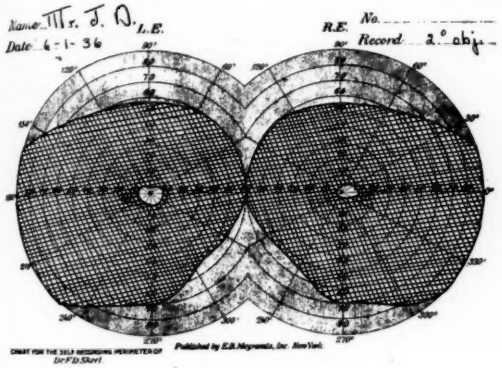

Fig. 1.

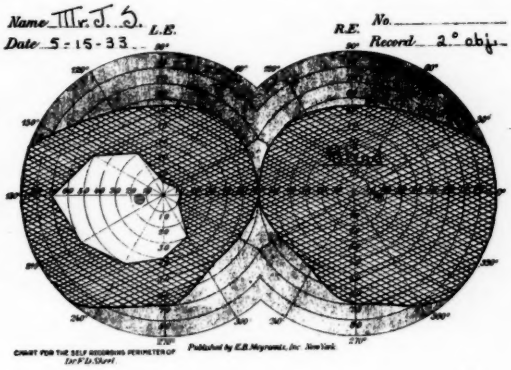

Fig. 2.

awakens to the fact that his vision is seriously impaired. Neglect of proper advice, and too great dependency upon miotics, causes irreparable damage from glaucoma quite as frequently as inability to obtain the glaucoma patient at a time when treatment will preserve vision. Even *acute* glaucoma is too frequently wrongly diagnosed as iritis; atropine treatment is given and irremediable damage results. I wish to offer a brief series of six cases each of which illustrates the technic of blindness from glaucoma, including delay in treatment, wrong diagnosis, refusal of operation, failure of follow-up treatment, and complications which are preventable.

Case Reports

Case 1.—Mr. J. D., aged forty-four, a myope, complained of gradual loss of sight in his right eye over a period of three years. No ophthalmoscopic examination had been made, glasses being changed from time to time by an optometrist. Vision in the right eye was 20/70, in the left eye 20/30. The fields of vision were reduced as shown in Figure 1. Tonometric measurements were 38 mg. and 43 mg. right and left respectively.

Diagnosis: Glaucoma simplex. Simple iris inclusion operations reduced tension which when last measured, February 15, 1936, was 9 mg. and 13 mg. repectively, the reduced fields of vision being maintained and the vision of the left eye remaining 20/30.

Comment: Had this man received a *competent* examination when vision began to fail, when optic nerve impairment would have been noted, he would have retained vision in the right eye and avoided marked loss of the visual field now reduced to "gunbarrel"

ophthalmologist, who recognized simple chronic glaucoma. He was properly advised as to treatment and periodic examinations. He neglected the advice and treatment prescribed until he became quite blind in the right eye, and the left eye began to fail. He again visited his ophthalmologist and was informed of the serious nature of his trouble. A miotic was prescribed, which was used intermittently. When he came for examination December 15, 1932, vision in the right eye was reduced to light perception; in the left eye to 20/50 with an hemianopic field of vision as shown in Figure 2. Tension in the right eye was 61 mg., left eye 55 mg., with the Gradle-Schiotz tonometer. Examination showed marked glaucomatous cupping and atrophy of the optic nerves.

Diagnosis: Right eye: Absolute glaucoma; left eye: Eye-advanced glaucoma simplex.

December 17, 1932, an iris inclusion operation was done to effect permanent subconjunctival drainage, reducing tension to 15 mg. This was maintained five months. A supplementary operation was required May 18, 1933, because of recurrent tension. Periodic tests over three years showed his remaining field of vision is preserved. His vision has remained 20/30.

Comment: The seriousness of the diagnosis and prognosis was not impressed upon this man until he had completely lost vision in one eye, and vision of the left eye reduced to 20/50 with marked field of vision changes. The "neglect" in this case may in part be ascribed to failure to actually frighten this patient into seeking relief from threatened blindness at the opportune stage. *Preventive* surgical treatment would have been more effective if begun several years earlier.

Case 3.—Mrs. J. M., aged sixty, came April 13, 1934. Vision had been failing for two years. Her family

doctor in June, 1933, made a diagnosis of cataracts. She was told to wait until vision was gone and then have her cataracts removed. Her physician did not examine her eyegrounds. When first seen vision was:

Right: 20/200 S. 150
Left: 8/120 No S.

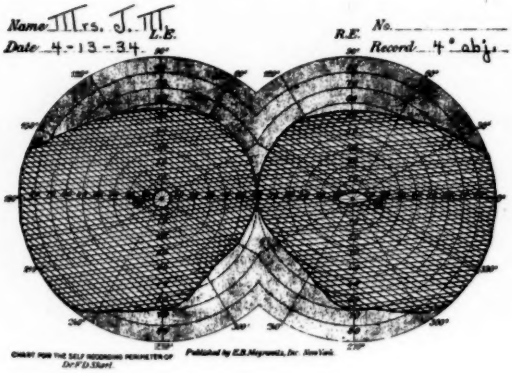

Fig. 3.

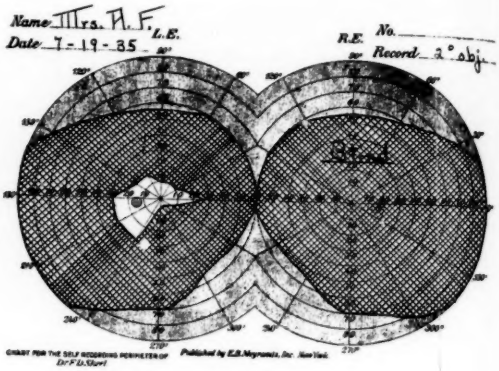

Fig. 4.

There was advanced glaucomatous cupping of both nerve heads. *There were no cataracts.* Tension: right 46 mg., left 29 mg. Fields of vision as shown in Figure 3.

Iris inclusion operations on both eyes were done April 18, 1934. On June 16, 1934, vision in the right eye was 20/30, in the left eye 20/50-2. Tension: right 17 mg., left 18.5 mg.

Comment: There can be no more striking comment upon this case than the illustration of the very great reduction of the fields of vision. Failure to make an ophthalmoscopic examination and ascertain that destructive glaucoma and *not* cataracts was the correct diagnosis. As is always the case, vision lost from glaucomatous atrophy of the optic nerves was permanent.

Case 4.—Mrs. A. F., aged sixty-six, came July 19, 1935, with the statement that the sight of her right eye had gradually been lost during a period of five years. She had consulted an oculist in 1934. He properly diagnosed her glaucoma and prescribed treatment. She consulted a second oculist, who confirmed the diagnosis, approved the treatment, and strongly advised its continuance. She continued it very irregularly, and for ten weeks preceding her visit to me had omitted it entirely.

On my first examination vision in the right eye was found reduced to hand movements, in the left eye to 8/100. She was unable to read any ordinary print. The tension of the right eye was 43 mg., of the left 61 mg. She had typical bilateral cupping of the optic discs. The field of vision could not be mapped out in the right eye; in the left eye reduced to the lower temporal quadrant as illustrated in Figure 4.

Tension could be reduced but not controlled within normal limits by the faithful use of miotics. Operation was emphatically advised. During the succeeding four weeks she would not consent to operation under any circumstances, although urged to have this done immediately at each of five visits. She did, however, use the miotics prescribed very faithfully over a period of twenty months but would not come for examination.

Finally on March 13, 1937, examination showed tension in the right eye was 74 mg., in the left 58 mg. The field of vision had encroached upon the fixation point as shown. In the right eye the iris was atrophic. There was a mature cataract with a secondary iritis and the cornea had begun to show degenerative changes.

On March 24, the right eye was submitted to a trephine operation, the left to an iris inclusion operation. On April 10, 1937, vision in the left eye was 20/100. The tension of each eye was well controlled. The result will doubtless remain.

Comment: This patient could have saved the vision in the right eye and very good vision in her left eye had she followed the advice given her when she consulted the first oculist in 1934. She certainly was sufficiently impressed with the seriousness of the malady and the probable outcome of neglect of treatment. Neglect on economic grounds was not justified, but the *mental* hazard of undertaking surgery caused this woman to delay treatment. Perhaps it could have been avoided had she been properly reassured and her mental fears dispelled.

Case 5.—Mrs. M. P., aged seventy-three, came November 20, 1935, with a history of failing vision for one year. Three months previously she consulted an *optometrist* who made a diagnosis of cataracts, gave her sixteen treatments with colored lights, and sold her two pairs of glasses which did not improve her vision.

When first examined by me, her vision in each eye was reduced to counting fingers at one foot. The tension in each eye was 74 mg. with the Schiotz-Gradle tonometer. There was very advanced glaucomatous cupping with a moderate lens haze. She had very little lens opacity. Fields of vision could not be recorded.

On November 25, bilateral iris inclusions with meridional iridotomies were done. Although there was no improvement in vision in either eye, the tension was reduced to 22 mg. in the right eye and 10 mg. in the left eye.

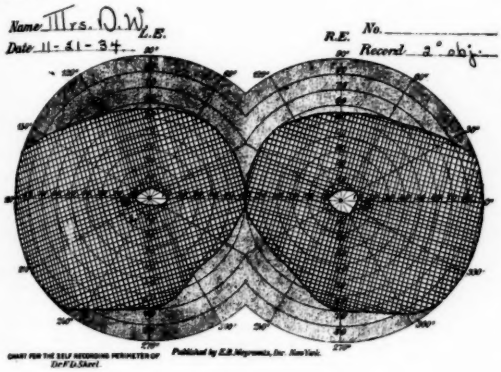

Fig. 5.

Comments: This is distinctly a case of blindness due to neglect, incompetent examination, wrong diagnosis and *treatment* by an optometrist, whose attitude toward the case could hardly be called less than criminal. Of course the patient's ignorance of things ophthalmic was a very large contributing factor. Consultation with an "optometrist" for false economic reasons led to total blindness.

Case 6.—Mrs. D. W., aged seventy, consulted me on February 27, 1934. Vision had been gradually failing for about four years. She had consulted two physicians in her home town, who told her that she had cataracts, and advised operation for their removal.

Examination showed cataracts present with vision 3/100 in each eye; *but* the projected fields of vision were exceedingly contracted in all directions to within 10 degrees of the fixation point. The pupils were moderately dilated. The tension registered 33 mg. in the right eye and 38 mg. in the left. The fundi could not be seen because of immature cataracts in each eye.

April 2, 1934, bilateral iris inclusion operations successfully relieved tension, which has remained well controlled. The lens opacities in each eye gradually increased. On May 18, 1934, the cataract in the left eye was successfully removed. August 27, 1934, the right eye was also successfully operated upon for removal of the opaque lens. When last examined in April, 1937, her vision was 20/50 in each eye. The field of vision, of course, was hopelessly contracted, as shown in Figure 5.

Comment: This demonstrates a true loss of fields of vision due to neglect in recognizing a glaucomatous state underlying cataract development. When she came for consultation, her visual fields were contracted to "gunbarrel" type. Although the glaucoma was successfully relieved, and the cataracts removed, the patient would have retained much better vision if an earlier diagnosis had been made.

These all too frequent instances of patients presenting themselves with marked loss of vision and visual fields vividly illustrate the results of neglect. Most of them were avoidable. Such cases plainly emphasize the necessity for careful study of the eyeground, measurement of intraocular tension and testing of the visual fields as a part of every eye examination.

Eye examinations should be a part of all physical surveys. There is no more important medical social work than that pertaining to the eye health of school children. No class of physical defects can be so effectively diagnosed or corrected at such little expense as errors of refraction. Even with the best possible correction of refractive errors and the most favorable school conditions, there is a large class of children whose vision is inadequate for ordinary purposes of education. In the three major cities in Minnesota in 1935-1936 we had 234 students enrolled in special sight saving classes. The selection of these students by our school hygiene directors deserves the highest commendation. Outside of these three cities, unfortunately, there are no school hygiene directors. The chief difficulty lies in the rural districts where too little progress has been made toward examination of the pre-school child. Every teacher should acquire knowledge and experience in vision testing technic. In the rural districts public health and school nurses should be especially trained in the examination and detection of the unusual defects of vision requiring special educational facilities. The medical profession must provide the leadership in the mobilization of effort toward conservation of the visually handicapped child, and in the education of parents as to the proper means of securing it. Newspaper publicity (untainted by commercial advertising), especially syndicated articles by competent authorities, are good propaganda. Likewise, articles in such lay journals as *Hygeia* and *Public Health* are good public education.

Since no sight saving classes are available for children in rural districts, parents should be better informed regarding the facilities afforded at our State School for the Blind. A State School for the Blind is quite generally considered an institution at which only children who have no sight can be educated in reading Braille or be taught some means of useful employment. As a matter of fact, our State Schools for the Blind

now embrace a much larger activity. In recent years every effort is being made to parallel the work of our city sight saving classes and to afford the opportunity for education of children from the state at large equal to that provided in our larger cities. Sight conservation classes are being constantly developed under a competent staff of instructors so that in all probability, with augmented enrollment of this special group, the name of the institution will ultimately have to be changed. This subject deserves emphasis because physicians and the public are not aware of the fine opportunities afforded and available to children from outside the larger cities.

Still another conservation opportunity deserves special mention: namely, children with monocular strabismus. They constitute the largest single group in which there exists serious impairment of vision in one eye. It seems necessary to emphasize three well established facts:

1. That strabismus is rarely outgrown.
2. That in 50 per cent of children crossed eyes can be straightened by means of proper and complete correction of refraction errors and with orthoptic training.
3. And, most important, that the neglected crossed eye child loses vision in the deviating eye unless measures are taken to correct the defect before the pre-school age. Vision once lost is rarely regained after the age of six.

Almost daily one is impressed with the fact that physicians, whose advice is first sought by parents of children with crossed eyes, do not properly appreciate the fact that this loss of vision occurs in the deviating eye and is avoidable. Amblyopia, or loss of vision in the deviating eye, results from involuntary suppression or inhibition of vision to prevent diplopia. If treatment can be begun and completed before school age, this amblyopia can usually be prevented, or vision can be reclaimed, by proper treatment.

It is remarkable how many parents bring their children for examination on account of strabismus only when they attain school age, or for esthetic reasons. In some manner they too frequently gain the impression that treatment is unnecessary and without prospect of benefit until the child is ready for school. When informed that the deviating eye has defective vision, they express great surprise and not infrequently resentment against earlier advice they have obtained.

By obtaining these children during the pre-school age, we are able to measure and correct the underlying errors of refraction, and, by occlusion of the straight eye, force the use of the deviating eye to prevent the gradual deterioration of vision which invariably develops. This conservation of vision in the deviating eye is often possible even before it is expedient to prescribe glasses. In the last few years, the new devices developed for the encouragement of fusion training and muscle exercises have been found very useful in competent hands. Optometrists sometimes capitalize the matter of muscle exercises to a point of absurdity and entirely disregard the true principles of orthoptic training. Unfortunately the gullible public, because of the unwarranted assumption of the title "doctor," does not discriminate in its choice between optometrists and ophthalmologists. It is impossible to examine a child's eyes accurately without the use of cycloplegia. Tests must be objective and not subjective. Proper examination of the child's eyes requires skill in retinoscopy. It cannot be properly done without the use of cycloplegics. The advice of the family physician in these matters should be final. He must emphasize the importance of early examination of the cross-eyed child; that effective prevention of deterioration of vision must be begun as soon as the strabismus manifests itself, or at the earliest age possible; and that a proper examination cannot be made without the aid of cycloplegia.

In conclusion: Prevention of visual impairment is not exclusively a problem confined to the ophthalmologist. It affects every branch of medicine. It concerns the obstetrician, neurologist, general physician, industrial surgeon, and the school's hygiene director. Examination of the eye is as much a part of a general health examination as that of any other part of the anatomy. When we are stressing the necessity and importance of periodic physical examinations, let us not forget the use of the ophthalmoscope and every other scientific aid for determination of the *reasons* for defective vision.

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PROTEIN DEFICIENCY EDEMA*

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EDEMA is one of the most common physical findings and occurs in a number of diseases. Often its cause may be most obscure and its successful treatment prove to be stubborn and puzzling. Edema has been defined by various clinicians and pathologists as "an abnormal collection of fluid within the tissue spaces and body cavities." Ordinarily it is our custom to think of cardiac and renal diseases primarily when we see a patient presenting edema, forgetting that edema may not only be cardiac or renal, but also mechanical or obstructive, inflammatory, allergic, and nutritive.

Factors entering into the development of edema are: (1) increased intracapillary pressure; (2) changes in the serum proteins; (3) increased capillary permeability, and (4) changes in the electrolytic concentration. Other factors, however, also enter into the formation of edema.

Starling[21] was probably the first to give a sound explanation of the pathogenesis of edema. He recognized that the capillary wall is a membrane permeable to certain substances including crystalloids such as glucose, urea, and salts, which easily pass through and exert no osmotic pressure; but that colloids, such as the serum proteins which do not normally pass through the wall do exert an osmotic pressure. It was his belief that the mechanical or hydrostatic intracapillary pressure at the proximal end of the capillary causes fluids to filter through the capillary wall into the tissue spaces and that at this end the capillary pressure is higher than the colloid osmotic pressure. At the distal or venous end of the capillary the colloid osmotic pressure is higher than the mechanical pressure and so fluids pass back into the capillary from the tissue spaces (Fig. 1). Landis[10] has demonstrated that normal colloid osmotic pressure varies from 29 to 40 cm. of water, with an average of 36 cm. of water[11] and that the hydrostatic pressure within the capillary varies from 45 cm. of water at the arterial end of the capillary to 22 cm. of water at the venous end. Knowing these figures and therefore being able to correctly visualize pressure relationships existing due to these two factors, it at once becomes apparent that there is a definite equilibrium existing between the

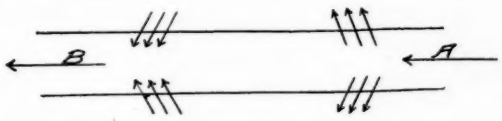

Fig. 1. Normally the hydrostatic pressure at the arterial end of the capillary **(A)** exceeds colloid osmotic pressure so that fluid passes through the capillary wall into the tissue spaces. At the venous end of the capillary **(B)** the colloid osmotic pressure exceeds the hydrostatic pressure and tissue fluid passes back into the blood stream.

hydrostatic pressure on the one hand and the colloid osmotic pressure on the other. Any increase in hydrostatic pressure, whether due to increased venous pressure from heart disease or mechanical obstruction from venous thrombosis or tumor masses, will result in an increased intracapillary pressure with the gradual accumulation of fluid in the tissue spaces, all other factors remaining equal. Likewise, a decrease in the colloid osmotic pressure will cause less tissue fluid to pass back into the capillary and edema will result. Trauma, warmth, and change in posture increase the intracapillary pressure[8,9,12,26] and anoxemia tends to cause an increase in capillary permeability.[7] It has also been demonstrated[14] that mechanical obstruction will result in increased capillary pressure and edema. If it were not for the pressure exerted by the tissues themselves[20,22] edema would soon result in people who are in the erect position for long, and indeed it frequently does in those individuals who are on their feet for long periods of time and in whom loss of tissue elasticity has occurred. This is even more common during hot weather, as we know, and in the tropics. The tissue fluids and particularly lymph may exert an osmotic pressure and it is an age-old belief that salt will produce edema, the sodium ion in particular.

Reviewing these various factors which exist in the pathogenesis of edema, Landis[11] has classified them into primary and contributory factors and

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shown the relationship existing between them and various clinical types of edema (Table I).

TABLE I. FACTORS IN THE PATHOGENESIS OF EDEMA
(after Landis)

Factors favoring edema formation	Clinical examples
A. Primary factors	
1. Elevated capillary pressure	1. (a) External pressure on veins (b) Thrombophlebitis (c) Cardiac edema with venous congestion
2. Lowered Colloid osmotic pressure	2. (a) Nutritional edema (b) Nephrotic edema (c) Cardiac edema (with malnutrition)
3. Damage to capillary wall	3. (a) Inflammatory edema (b) Nephritic edema (c) Cardiac edema (chronic anoxemia)
4. Lymphatic obstruction	4. (a) Lymphedema (b) Cardiac edema with venous congestion
B. Contributory factors	
5. Low tissue pressure	5. Edema of periorbital tissues and genitalia
6. High salt intake	6. Increases edema if water is available
7. High fluid intake	7. Increases edema if salt is available
8. Warm environment	8. (a) Heat edema (b) Increases all types of edema
9. Disturbed innervation	9. (a) Trophedema (b) Unilateral edema in hemiplegia

It is the edema which is caused by a reduction in the colloid osmotic pressure and in particular that termed nutritional edema, but more aptly called protein deficiency edema, which I now wish to discuss.

Numerous terms have been applied to this form of edema and included among them we find such names as nutritional edema, starvation edema, war edema, prison dropsy, edema from inadequate food, and edema without albuminuria. Since all these forms of edema are associated with a common factor, i.e., a reduction in the serum proteins in the blood, the blanket term of protein deficiency edema seems to be the most applicable.

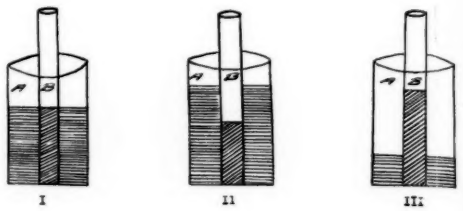

Fig. 2. All other factors being equal, in (I) the serum proteins are normal and the fluids are in a state of equilibrium; in (II) the serum proteins are reduced and the fluid collects in the tissues; in (III) the serum proteins are increased and the tissues are dehydrated. **A**, Tissue spaces. **B**, Capillaries.

Although it has been only during the past two decades that our attention has been concentrated on this form of edema, in reality its history dates back for centuries. Nixon,[19] quoting Herrick, states regarding the army besieging Naples in 1528—"Those soldiers who were not already confined to bed in their tents, were seen with pallid visages, swelled legs, and bloated bellies, scarcely able to crawl." This was probably the first description of edema due to a deficiency of protein. Again it was noted in the men of Napoleon's Army on the retreat from Moscow in 1812. In 1865, Cornish referred to its treatment. During the Great War, the so-called "starvation edema" appeared at Lillie in 1914. From 1915 on there were numerous cases in the German prison camps, Galacia and Poland.

Starling's basic work has been referred to above. According to his conceptions, any disturbance in the equilibrium existing between intracapillary pressure and colloid osmotic pressure would lead to the appearance of edema, so that with a reduction in serum protein concentration, edema would appear (Fig. 2). Since then a great deal of experimental and investigative work has been done on animals and humans. Leiter[15] produced edema in dogs by repeated plasmapharesis. Weech and Ling[23,24] in China, successfully demonstrated in the undernourished Chinese the relationship between the appearance of edema and the existant serum proteins. You-

mans,[27] in this country, has shown that the epidemic edema existant in Tennessee is definitely related to a deficient diet and thereby to a lowered serum protein.

Moore and Van Slyke,[18] averaging their values with those of other authors set the normal total protein at 6.3 to 8 grams per cent, serum albumen at 3.6 to 5 grams per cent and serum globulin ranging from 2 to 3.5 grams per cent. The critical levels, that is the levels at which edema appears, were set at 5.5 grams per cent for the total protein and 2.5 grams per cent for the serum albumen. Weech and Ling[24] felt that edema appeared when the serum albumen fell to 2.9 grams per cent. Weech[23] has shown that instead of the normal albumen globulin ration of 1.3 to 2.5 the ratio is reversed in nutritional edema, although not as low as in nephrotic edema. Youmans[25] feels that edema may appear when the serum protein has not yet fallen to critical levels, but is at a low normal reading and attributes the appearance of edema to such factors as the salt and water intake, posture, environmental temperature and the pressure or tension exerted by the tissues.

Of the two types of protein playing a rôle in the production of protein deficiency edema, albumen, and globulin, we find that the former is of the greater importance. Goeverts[5] has shown that serum albumen possesses an osmotic pressure of 7.54 cm. of water, while serum globulin exerts a pressure of 1.95 cm. of water. It is obvious therefore that, of the two, serum albumen is of the greater importance because it exerts an osmotic pressure four times that of the serum globulin. We must remember in discussing this type of edema that it is the osmotic pressure which is of importance and that since albumen exerts the greatest pressure any reduction in this protein fraction will be of more importance and of greater effect than reduction in the serum globulin. This has been definitely shown by the various authors mentioned above. Another factor which makes the loss of albumen of greater importance than globulin loss is that the albumen molecule is smaller than the globulin molecule and consequently more easily lost.

For many years although the level of serum proteins was definitely associated with the appearance of edema, it has not been until more recent years that this has been recognized in cases other than the so called nutritional edema. We now are able to recognize various groups of this form of edema namely due to:

1. Deficient protein intake
2. Loss of protein
   Proteinuria (Albuminuria)
   Diarrhea
   Hemorrhage, especially from the bowel
   Purulent drainage
   Transudates with a high protein content
3. Inability of the body to assimilate protein.

The production of edema by diet deficient in protein has been dwelt upon at length. The works of Youmans, Weech and Ling, and Liu have been noted. Landis and Leopold[13] have noted the appearance of edema in a patient with tuberculous enteritis and a massive diarrhea. Meyer,[17] Eaton and Jones[4] and Menard[16] and others have all noted the appearance of edema in patients when the protein intake has been deficient, and there is a concomittant loss of protein by drainage together with a high fluid and salt intake. Binger and Keith[3] have observed edema associated with a deficiency in serum proteins due to inability of the individual to assimilate protein because of severe pancreatitis and hepatitis.

To clarify what has been said I now wish to report two cases.

The first is that of a white female, aged forty-seven, who was first seen in a state of cardiac decompensation. Her history was primarily that of thyrotoxicosis with the subsequent development of thyrotoxic heart disease. Ligation of the superior thyroid arteries had been performed in an attempt to control the toxicity. However, cardiac failure continued with increasing edema, dyspnea, and nausea. Examination revealed findings typical of a hydrothorax on the right side, a heart enlarged to the left with a systolic murmur at the mitral and aortic areas, both well transmitted. The pulse rate was totally irregular and the blood pressure 134 systolic. The abominal wall was slightly edematous and the liver was palpable three centimeters below the right costal margin. There was massive edema of the lower extremities. The venous pressure was 12 cm. of water. Laboratory examination included urinalysis, blood count, and blood chemistry, all of which were normal.

The patient was placed at absolute bed rest, fluids were limited, oxygen was supplied by nasal catheter at 6 liters per minute and the patient was digitalized. For forty successive days a thoracentesis was performed on the right side with the daily withdrawal of 1,000 c.c. of fluid. Despite these measures she failed to improve. The edema of the abdominal wall in-

creased until it was massive in extent. Theocin was given in ten grain doses three times a day without success. Salyrgan preceded by ammonium chloride was of no avail. Repeated urinalysis revealed only a small quantity of albumen. Finally an estimation of the protein in the thoracic fluid was performed and was found to be 37.5 grams per liter. The serum protein was 5 grams and the serum albumen 3.2 grams with the globulin 1.8 grams. The albumen globulin ratio was 1.7. It was thought that a protein deficiency existed and the patient was consequently given 150 grams of protein in her diet daily. She rapidly grew better and was shortly able to be out of bed, have metabolic rate determinations and successfully received deep x-ray treatment of the thyroid.

We have here a patient who was thyrotoxic and who eventually developed a thyrotoxic heart disease and then decompensation. Coincident with her state of cardiac decompensation and increased intracapillary pressure, she developed an increased capillary permeability probably due to chronic anoxemia and thus a passage of the protein through the capillaries into the body cavities. We find that hydrostatic or intracapillary pressure, capillary permeability, and the subsequent loss of serum protein plus the possibility of malnutrition resulted in anasarca. A vicious cycle had been instituted which could be corrected only by increasing the serum protein through increasing the protein in the diet.

The second case I wish to discuss, I present to you mainly to demonstrate that with adequate clinical laboratory study unavailable, a diagnosis of protein deficiency edema may be made by thorough history taking, complete physical examination and simple laboratory data.

The patient was a white female, twenty-six years of age. She offered as a chief complaint swelling of the ankles, about the eyes, and the skin of the chest and abdomen. This had been present off and on for about six months and apparently had no diurnal occurrence. It had not handicapped her, but had been a source of worry. History by systems was essentially negative. Physical examination revealed no evidence of cardiac, renal, or hepatic disease. There was pitting edema of the ankles. Laboratory data included a complete blood count which was entirely normal and gave no evidence of anemia. Repeated examination of the urine, using a twenty-four hour specimen, consistently failed to show any albumen. The cause of her edema was difficult to explain. I have purposely failed to mention her social history which indicated that for two years she had worked in a packing plant and having sickened of the sight of meat had included very little in her diet. She had also consumed very little milk, cheese, or eggs. Her diet consisted mainly of fruit and vegetables. It was impossible to perform accurate scientific laboratory tests, but it was my feeling that this patient had a protein deficiency edema. The situation was carefully explained to her and she was urged to eat a certain amount of meat, milk, eggs, and cheese daily and to limit the amount of salt. She followed dietary instructions faithfully and has since had no recurrence of the edema.

This is satisfactory evidence that we do not need to resort to the extremely well equipped laboratory, but may depend upon our history and physical examinations plus simple data which may be secured in any office.

Meyer's[17] case demonstrates the effect of protein restriction, protein loss by purulent drainage, and the dilution of serum proteins by the intravenous administration of large amounts of saline and glucose. It bears out the contention of Beard and Blalock[1,2] that in surgical cases the administration of intravenous fluids dilutes the serum proteins, so that already lowered they may be decreased still farther with edema resulting.

The treatment of protein deficiency edema naturally consists primarily in the administration of protein in large quantities. However, water without salt cannot be retained. In many cases when the serum proteins are at the borderline, that is when they are at or slightly above the critical level, edema will appear.[25] In these cases the simple restriction of salt from the diet will result in the disappearance of edema. Therefore in treating this type of edema, it would appear best to not only give high protein diets, but also to make that a salt free or salt low diet with restriction of fluids. The protein should be given as high as one hundred to one hundred and fifty grams daily. Animal protein will secure more rapid and satisfactory results, and the control of salt and water intake with bed rest are advisable. Scientifically it is possible to compute the amount of protein necessary by giving one gram of protein per kilogram body weight plus that which is lost in the urine, if that is the route of protein deprivation. Blood transfusions and the intravenous administration of gum acacia, after the method of Senn and Hartman[6] are to be resorted to when nothing can be taken orally. If small amounts of liquids can be tolerated by mouth, egg albumen, gelatine, and milk are the proteins of choice. The use of mercurial diuretics is questionable and certainly one should

defer their use if there is the slightest question of already existent renal damage. Ammonium chloride and other salts may be used in an attempt to upset the electrolytic concentration and thus produce diuresis.

A word may be said here relative to the edema existent in cardiac and renal disease. In the former, particularly the hypertensive type, the patients are often given low protein diets, thus lowering the serum proteins and producing a potential hydropigenous state. This is often kept up after the appearance of edema. It would be better to give these individuals diets high in protein. If this is done, one is less apt to find resistant and intractable cardiac edema. Again, the same may be said regarding the nephritic. Here there exist all of the forces having to do with the production of edema. It would be more advisable in these individuals, if the protein end products in the blood are normal, to give diets containing an adequate amount of protein. In many operative cases the nutrition has been poor and the serum proteins are low. Post-operatively the patient is given copious amounts of saline, often with the appearance of edema. Knowing that these fluids may dilute the proteins, lowering them still more, when because of poor nourishment they are already lowered, and that salt in these cases may produce edema, it would seem best to maintain the water balance by giving glucose in isotonic solution.

Therefore, we see that the essential treatment of protein deficiency edema is to give high protein, salt free diets, and that the administration of protein in other types of edema is an aid in decreasing the same. In conclusion, it is to be remembered that edema is a symptom and sign, not a disease, to be interpreted as part of a disease process with the treatment incorporated in that of the original disease.

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THE SERUM TREATMENT OF PNEUMOCOCCUS LOBAR PNEUMONIA*

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THE problem of pneumonia has been known since the time of Hippocrates. It has been reported in all countries, and in all climates. It attacks the rich and poor, the young and the old and is especially prevalent and fatal in the elderly. The mortality has always been high and the outcome unpredictable.

Nothing was known of the bacterial etiology of pneumonia until the year 1880. In September of that year, Sternberg in Germany inoculated rabbits with his own saliva and isolated a micrococcus. About the same time Pasteur in Paris discovered a similar organism in the saliva of a child dead of hydrophobia. There was, however, no suspicion that this organism was concerned in the etiology of pneumonia.

It was not until 1884 that Fraenkel in Germany determined that the organism found by Sternberg and Pasteur in saliva and known as the "coccus of sputum septicemia," was the most frequent organism in acute pneumonia. At first there was some confusion between this organism and that described by Friedlander in 1883.

By 1886, Fraenkel and Weichselbaum had demonstrated the micrococcus in most cases of pneumonia, and later studies caused their organism to be accepted as the sole cause of acute lobar pneumonia. Their organism was described as elliptical or lance shaped and was found to occur in pairs; hence the designation—diplococcus. A capsule was always found surrounding the organism, and this served to differentiate it from the streptococcus.

Soon attempts were made to produce an immune serum. The brothers Klemperer, from Leyden's Clinic in Berlin, were able to immunize animals by the injection of filtered bouillon cultures of pneumococci obtained from patients ill with pneumonia, and to use the serum from these immunized animals to immunize other animals, or cure them when they had been previously infected. They attempted to produce immunity in human cases of pneumonia by the injection of the blood serum from patients convalescent from pneumonia, and they reported favorable results in six cases. But the actual obtaining of an effective anti-pneumococcic serum was to be long deferred, as a review of the literature for the past fifty years discloses.

In his textbook on "The Practice of Medicine," in the 1901 edition, Osler has this comment to make on serum therapy:

"Many trials have been made of the curative value of anti-pneumococcic serum in the treatment of pneumonia. Thus far it has not been shown that this influences in any marked degree the course of the disease in man."

In 1902, Neufeld observed, while working with the pneumococcus, that when a suspension of pneumococci was mixed on a slide with homologous immune serum, a reaction occurred which was characterized not only by agglutination of organisms, but also by a "quellung" or swelling of their capsules. He claimed that the reaction could only be observed in the moist state without fixation.

He attempted to apply this method to the typing of pneumococci. His method was to obtain a drop of peritoneal exudate obtained from the mouse's peritoneum six to eight hours after injection, mix this with diagnostic serum and a drop of dilute crystal violet, cover with a cover slip, and examine under oil immersion. He could type the organisms in many instances, but because of rapid drying he abandoned this method for the macroscopic agglutination method.

Neufeld found considerable variety in pneumococci obtained from different patients ill with pneumonia, a variation which he referred to as races or strains. He found one rather predominant strain and succeeded in producing an immune serum by injecting a horse with cultures of this strain. He found that there were a considerable number of strains of pneumococci that would not agglutinate with his immune serum.

In 1910, the Rockefeller Hospital for Medical Research was opened in New York City, and Rufus I. Cole, who had formerly worked with

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Osler, was put in charge of the pneumonia ward. Apparently Cole remembered Osler's dictum that "Pneumonia is a self limited disease and runs its course uninfluenced in any way by medicine," and set himself the goal of finding some specific therapy to lower the death rate in pneumonia. Just as Osler gathered around himself an inspired group of research men and teachers, so, as one reviews the literature on pneumonia, one gathers the impression that Cole has done the same thing.

In the year 1913, there appeared a paper by Dochez and Gillespie[6] which, as far as I can find, was the first published work from the research study carried on in the pneumonia wards of the new Rockefeller Hospital. They noted that they had received some immune anti-pneumococci horse serum from Professor Neufeld which they called Serum No. 1, and with this as a standard they studied the pneumococci obtained from human cases by means of protection experiments.

As a result of their study they were able to separate and classify pneumococci into four groups which they designated by the Roman numerals I, II, III, and IV. They stated, "A study of cross protection and agglutination among members of Group IV indicates that this group comprises a number of distinct varieties of the pneumococcus which can not be related to one another by immunologic reactions. Culturally, they are true pneumococci and manifest all the common characteristics of the pneumococcus. A large majority of all the strains studied fall into the three fixed Groups I, II, and III. The minority fall into the mixed or heterogeneous Group IV. The individual members of Groups I and II can be distinguished from each other only by highly immune reactions. The heterogeneous Group IV seems to consist entirely of isolated individuals."

It is interesting to note that just twenty-four years ago at a meeting of the American Medical Association in Minneapolis in June, 1913, Rufus I. Cole[2] reported on the specific treatment of seventeen cases of pneumonia in the Rockefeller Hospital. Of these, fourteen were of Type I, and three of Type II, as based on the new classification of Dochez and Gillespie.

His method of treatment was as follows: On admission to the hospital a blood culture was obtained, and also a specimen of sputum, and if this was not obtainable, a culture was made from the lung by puncture. A portion of sputum, if obtained, was injected into the peritoneal cavity of a mouse. After five hours the peritoneal cavity was washed out with salt solution, the solution centrifugalized, and a suspension of cells was obtained. Agglutination tests were then carried out against known typed sera. If the sputum organisms failed to be agglutinated by Type I or II sera, it was considered useless to undertake the serum treatment.

While his study of type differentiation was in progress, 2 c.c. of horse serum was injected subcutaneously into the patient to determine the presence of horse serum sensitization. If the patient was found to be non-sensitive, an amount of specific anti-pneumococcic serum, varying from 50 to 100 c.c. and diluted with 50 per cent normal saline solution, was injected intravenously. The response of the patient served as a guide to subsequent treatment. Usually the dose was repeated not oftener than every twelve hours. The amount of serum used ranged from 190 c.c. up to 460 c.c. The earliest the treatment was begun was on the third day of the disease, and the latest was on the sixth day of the disease.

Of the fourteen Type I cases, only one died, a mortality rate of 7 per cent. Of the three Type II cases, one died, a rate of 33.3 per cent. In the surviving cases the serum seemed to have a favorable effect in lowering the temperature, in lessening signs of intoxication, and in shortening the course of the disease. Cole deduced from his studies that specific treatment with specific anti-pneumococcic serum was valuable, but that its use should be begun early in the course of the disease before the infection had reached the maximum degree, beyond which no amount of serum appeared to be effective.

The next step in the further understanding of the bacteriology of the pneumococcus came after the pandemic of influenza of 1918-19, with its terrific mortality from pneumonia. The need seemed greater to find better serum therapy. The Metropolitan Life Insurance Company appointed an Influenza Commission which worked in conjunction with the Bureau of Laboratories of the Health Department of the City of New York.

In 1921, Cooper and her associates[3] obtained cultures from the nose and throat of 138 patients

with rhinitis and pharyngitis, from simple influenza-like cases, and cases of influenza complicated with broncho-pneumonia. In forty-seven instances their cultures were sufficiently pure for further study. They found no single variety of pneumococcus to dominate in the inflammatory conditions studied, and they concluded that the pneumococcus was not a primary etiological agent in the contagious type of influenza.

Apparently these authors continued to work on the different types of pneumococci, for in the year 1929 Cooper and her associates[4] announced that they had been able to subdivide Group IV into ten sub-groups which they designated as Groups IV-XIII.

Three years later, Cooper and her associates[5] stated that "An investigation into the possibility of treating cases of pneumonia with specific antisera (other than Types I and II antisera) was undertaken because clinical results indicated that cases of pneumonia benefited slightly, *if at all*, by heterologous antisera. For the purpose of preparing suitable antisera from this heterogeneous Group IV, it was first necessary to further classify the various types in this group; to prepare diagnostic antisera for their type identification, and then to prepare specific antisera for treatment." As a result of this study they claim to have identified nineteen additional strains or types which were classified as Types XIV to XXXII.

In their plans to prepare new specific antisera for these newly identified types, they abandoned the old measure of bulk for use in gauging dosage of antisera and substituted the measure of the unit in order to standardize their serum. They define a unit as follows: "A unit is ten times the smallest amount of antiserum that protects the majority of mice against approximately 100,000 fatal doses of culture." They selected 500 units per c.c. as a minimum strength for therapeutic antisera for Types I and II and were attempting to produce antisera of equal potency from their newly identified strains.

In conclusion the authors stated that exclusive of the known Types I, II, III, in lobar pneumonia of adults, Types IV, V, VII and VIII predominated; whereas in the pneumonia of children, Types V, VIa and XIV predominated. Fourteen of the types were found in pneumonic meningitis, Type XVIII being found most often.

Since type identification of the pneumococcus had come to be such an important laboratory procedure, it was necessary that a simple, rapid, and practical method be found. Many methods of pneumococcus typing had been devised, but the one most commonly used was that method in which the peritoneal exudate, obtained from the mouse 18 to 24 hours after injection of the sputum, was tested for agglutination with various typing serums.

In the year 1930, Sabin[10] of New York described the method of injecting sputum into the peritoneal cavity of a mouse. At the end of five hours he withdrew some of the peritoneal exudate, mixed this on a microscopic slide with typing sera, stained with fuchsin, and examined for agglutination under the oil immersion. He stated that agglutination never occurred with normal horse serum, or with heterologous serum.

In the year 1933, Sabin[11] recounted the fact that he had previously tried to use the "quellung" reaction described by Neufeld for type determination, but had been unsuccessful; however, a Dr. Goodner of the Rockefeller Institute, traveling in Germany, had observed the method of "rapid-sputum slide-type-identification" as it was carried out in Professor Neufeld's laboratory in Berlin, had used it successfully himself, and demonstrated it to the author. After several trials in the laboratory of the Bellevue Hospital, Sabin found that the test was not always reliable because of the fact that the horse anti-pneumococcus-serum commonly used for typing, frequently gave non-specific reactions. In order to overcome this difficulty he prepared anti-pneumococcic rabbit serum from the various known strains, and when the rabbit serum was used for serum typing, the results were uniformly specific. He found that Neufeld's crystal violet did not always stain the bodies of the pneumococci, so he substituted alkaline methylene blue stain, and this always stained the bodies of the pneumococci a dark blue, leaving clearly visible the swollen unstained capsule. Furthermore, he stated definitely that the sputum should be typed within one to two hours after it was coughed up because the pneumococci autolyzed very rapidly, and if the sputum was allowed to stand, they might completely disintegrate.

When in certain adults and most children,

sputum cannot be obtained by coughing, then the throat is swabbed, and the swab incubated in glucose broth for three to four hours. Then 5 c.c. of this culture is injected into the peritoneum of the mouse, the peritoneal exudate is withdrawn after five hours, and the microscopic "quellung" reaction can be carried out.

Sabin's capsule swelling identification method can be described as follows: "Two small flecks of sputum *coughed up from the lung* were placed on a large cover slip with a small platinum loop 2 mm. in diameter. To each bit of sputum an equal quantity of undiluated rabbit serum of known type was added, Type I serum to one, Type II serum to the other, and to these separate mixtures was added a loopful of standard alkaline methylene blue stain. A deep, large, hollow ground slide, big enough to cover both drops, was used. The edges of the slide were smeared with petrolatum and the slide placed over the cover slip and the preparation inverted and examined with an oil immersion lens and an artificial blue light."

Two minutes' time was allowed for the reaction to be complete. Sabin says it is important to remember that in the ordinary hanging drop preparations, pneumococci show no capsules although occasionally a faint halo of light without any definite outline may be seen about the organism. However, he continues, in the type specific mixtures of sputum and rabbit serum, one finds the pneumococci surrounded by a peripheral zone of characteristic appearance and distinct outline. The peripheral zone consists of a refractile substance which does not take the stain and has a ground glass appearance, the body of the organism within being stained deep blue. While agglutination does occur, Sabin says that agglutination is not the diagnostic reaction, but rather the swelling of the capsule, the so-called "quellung" reaction of Neufeld. So characteristic is this "quellung" reaction that the finding of only a single pneumococcus which shows this "quellung" reaction, is sufficient evidence on which to base the type of pneumococcus in the sputum. When Sabin checked his method against the mouse inoculation method, he stated that in not a single case of lobar pneumonia in which Type I or Type II pneumococcus was identified by the mouse inoculation did he fail to find the "quellung" reaction by his direct sputum method.

The chief value of Sabin's new type identification method is that it can be done as soon as the patient enters the hospital. This allows the institution of specific anti-pneumococcic therapy far sooner than was possible before. Sabin reports on six patients who come to the hospital twenty-four to thirty hours after the onset of their pneumonia. In each case Type I pneumococcus was the infecting organism, as revealed by his rapid typing method, and in each case serum therapy was spectacularly effective.

It is necessary now to trace the development of anti-pneumococci serum up to the present refined and concentrated preparation. In the year 1910, Cole received from Professor Neufeld a culture of a pneumococcus that was most frequently recovered from the sputum of patients ill with pneumonia, and from which Neufeld had prepared an immune serum. Cole and his associates inoculated horses with this Neufeld culture and developed an immune serum of their own which Cole designated as Serum No. 1. Any patients ill with lobar pneumonia in the wards of the Rockefeller Hospital whose sputum pneumococci were compatible with Cole's No. 1 serum, received treatment with No. 1 serum. Later, Cole and his co-workers produced a serum for infections with Type II pneumococcus. The results of serum therapy at the Rockefeller Hospital were reported in the year 1913, as noted earlier in this paper.

Cole used a crude unrefined serum, and the dosage was measured in cubic centimeters. Because of some anaphylactic reactions during the serum administration and the occurrence of serum sickness in a considerable number of treated cases, attempts were made to refine the serum and to concentrate it. Avery, in the year 1915, attempted to fractionate the proteins in the serum and to determine which of the proteins possessed the effective principle. By means of a 43 per cent ammonium sulphate solution, he was able to isolate the effective principle, but during the concentration of the serum there occurred a large loss of the effective substance, upwards of from 25 to 50 per cent, and the use of Avery's concentrated serum was not generally adopted.

In the year 1921, Huntoon, by a different method, concentrated large amounts of anti-pneumococcic serum, and his preparation was given an extensive clinical trial. But the reactions

PER CENT OF MORTALITY IN RELATION TO AGE AND TYPE (1,867 CASES)

Type	Under 20		20-30		30-40		40-50		50-60		Over 60	
	Cases	%	Cases	%	Cases	%	Cases	%	Cases	%	Cases	%
I	59	8.4	187	16.0	179	17.8	110	21.0	60	26.6	32	31.2
II	16	18.7	89	17.9	91	38.4	84	53.5	60	45.0	22	63.6
III	8	12.5	33	15.1	62	29.0	63	44.4	43	69.7	54	59.2
IV	51	13.6	142	13.3	155	21.0	136	29.4	76	38.1	55	54.5
Total	134	11.1	451	15.5	487	24.2	393	34.8	239	42.7	163	52.7

from the use of Huntoon's serum were violent, and the therapeutic results were not encouraging; therefore the use of this preparation was abandoned.

In the year 1924, Felton,[7] working in the Department of Preventive Medicine and Hygiene of the Harvard Medical School, stated that while studying the virulence of pneumococci and the difference in growth of pneumococci in normal horse serum and immune serum, he found the protective factor to lie in the *globulin* radical of the immune serum.

This globulin factor he was able to precipitate out by treating the serum with fifteen volumes of distilled water, later redissolving the precipitate in sodium chloride. The serum thus concentrated contained 30 per cent less protein and was concentrated from two to twenty times. This serum was administered to more than sixty cases of lobar pneumonia in the Boston City Hospital with favorable results.

Apparently this serum of Felton's did not prove entirely satisfactory because in the year 1928 Felton offered a new method[8] of serum concentration to replace the Avery method, which latter was found to produce a chill reaction when administered to patients ill with pneumonia. Avery's precipitating vehicle was 44 per cent ammonium sulphate. Felton substituted a 20 per cent solution of anhydrous sodium sulphate for the ammonium sulphate and achieved an equal concentration of serum. This newly concentrated serum of Felton was submitted for clinical use to the physicians of Boston who were intensively studying the serum treatment of lobar pneumonia.

With true Bostonian conservatism Felton appears to have waited for clinical proof of the efficacy of his serum, because in the summary of his paper of 1928 we find his deductions as follows:

"The serum concentrates from the water dilution method and the sodium sulphate method have been used clinically (in cases of lobar pneumonia) for the past two years, with only a small percentage of troublesome reactions; whereas the serum produced by the ammonium sulphate method, when used clinically, produced severe reactions in a high percentage of cases."

It is now nearly fifty years since that first attempt to produce an immune serum was made by the Brothers Klemperer in Berlin. But only within the past few years has the serum been perfected, the types of pneumococci sufficiently classified, and methods developed for immediate typing, so that serum therapy can be instituted early in the course of the disease.

General Considerations and Selection of Cases for Specific Serum Therapy

The most frequent types of the pneumococcus encountered in lobar pneumonia are Types I, II, III, VIII, VII and V, in approximately that order. These six types are found in the sputum in about 80 per cent of cases of lobar pneumonia. The mortality rate varies with the different types, but rises progressively in all types with advancing years, and is especially high after sixty years of age.

The above table, taken from an article by Cecil, Baldwin and Larsen,[1] shows the mortality and incidence of the various types of pneumonia according to age periods. In this table, Type IV includes Types IV to XXXII, inclusive, under the newer classification.

The mortality rate also varies with the absence or presence of bacteremia. The following table from the same source reveals this fact very dramatically:

INCIDENCE OF POSITIVE BLOOD CULTURES IN RELATION TO TYPE AND OUTCOME

	PATIENTS RECOVERED			PATIENTS DIED		
Type	No. Cases	Bl. Culture Positive	Per Cent Positive	No. Cases	Bl. Culture Positive	Per Cent Positive
I	87	9	10.3	38	24	63.1
II	39	3	7.6	36	28	77.7
III	17	2	11.1	19	8	42.1
IV	64	1	1.5	26	14	53.8
Total	267	15	7.2%	119	74	62.1%

Lord and Heffron[9] analyzed the percentage distribution of the various types of pneumococci in 9,639 cases of lobar pneumonia collected from the literature as follows:

Type I..... 33.4%
Type II 23.3%
Type III 8.9%
Type IV (all other types)..... 34.4%

This table shows that Types I and II make up nearly 60 per cent of all cases of lobar pneumonia in which the type of pneumococcus was determined. Certain general facts regarding these types are now available. Only lobar pneumonia due to Types I and II, and possibly Type VII, respond favorably to specific antipneumococcic therapy. Pneumonia due to these types is almost always primary and characteristically typical lobar pneumonia.

In atypical cases the physical signs may be uncertain for a time, or remain so throughout the illness. It is in such atypical cases, or in those with slowly developing physical signs, that early sputum typing clinches the diagnosis and clears the way to *early* specific serum therapy at a time when such therapy is especially effective.

With typical onsets in *male* patients, Types I or II pneumococcus infections may be expected in 75 per cent of the cases.[11] As far as children are concerned, it is difficult to obtain sputum for typing in children under twelve years of age. Owing to the low death rate, the small size of the veins, the difficulty of making repeated intravenous injections, and the lack of any conclusive results thus far obtained in children, treatment with serum is seldom attempted. Patients who are very old and infirm, those with pulmonary edema, and those already in a desperate state of illness, or in collapse, stupor or coma should not be subjected to serum therapy. Such patients are likely to die before adequate dosage can be given, and even a mild reaction from alien serum may hasten the fatal termination. An illness of such severity is not often encountered within the first four days of the onset.

Precautions Prior to Administration of Serum

According to statistics from the Massachusetts Pneumonia Study,[11] extending from the year 1931 through the year 1935, 936 cases of lobar pneumonia received treatment with specific antipneumonic serum. Of these cases, death was attributed to the serum in four instances, three due to thermal reactions and one to an allergy. In interpreting these statistics it is well to remember that a majority of these cases were in hospitals and under the observation of skilled physicians and carefully trained assistants.

The reactions from the serum are not related to its curative properties, but to the proteins in the horse serum. It is generally conceded at the present time that approximately 10 per cent of the population have either latent allergic tendencies or actual manifestations. Therefore patients who are to receive treatment must be selected with extreme care, and the details of serum administration followed out exactly as prescribed.

There are three known groups of patients to whom the administration of horse serum may be dangerous. The first and most hazardous are those with a history of hayfever, asthma, hives, angioneurotic edema, or eczema. The second and less hazardous comprises those who have had a previous injection of horse serum, recent or remote. There is a third group in which the history is negative for allergic diseases or previous injection of horse serum. The only means of detecting cases in this group is by the use of both the skin test and eye test. There is also the group of patients who develop sneezing attacks with watering eyes and running nose when near

horses. Such patients may also be sensitive to horse serum and *should be excluded from specific serum treatment.* Therefore before instituting serum treatment, obtain a history as to the above, and always make skin and eye tests with normal serum in every patient in whom serum administration is contemplated.

Methods for Making the Tests for Sensitivity

For a preliminary test plain horse serum should be no stronger than 1-100; i.e., one part of serum to 99 parts of physiologic solution of sodium chloride. The intracutaneous skin test consists of the injection of 0.05 c.c. of the diluted serum *between* the layers of the skin, using a tuberculin syringe and a fine (26-gauge) needle so that a small wheal is raised. A positive reaction consists of an urticarial swelling with central blanching, peripheral erythema, and itching. This occurs in five to twenty minutes.

By the eye, or ophthalmic test, is meant the instillation of one drop of the diluted plain horse serum into the conjunctival sac of one eye, leaving the other eye for a control. A positive reaction occurs within a few minutes. It consists of a slight injection of the conjunctival vessels and itching if the response is mild; and if the reaction is severe, marked injection of the vessels and edema of the conjunctiva and lids occur. The test should be made first with a 1-100 dilution. Only if this is negative, should a 1-10 dilution be tried. In case of a marked conjunctival reaction, instill one or two drops of 1-1000 adrenalin solution into the sac. *The ophthalmic reaction is generally considered a more true indication of serum sensitivity.*

If there is no history of allergy and the intracutaneous test with 1-100 serum is negative, a test should be made with a 1-10 dilution of serum. If the latter is negative, one may proceed with the administration of the serum.

The concentrated antipneumococci serum causes positive intradermal tests in a large proportion of normal persons and is unreliable for the determination of skin sensitivity.

It is well to make a control intradermal test with physiologic salt solution into the skin of the other forearm. Before any tests for sensitivity are made, fill a syringe with 1-1000 adrenalin solution and have it ready for use.

Should any doubt arise as to the interpretation of the tests for sensitivity, a preliminary trial injection of serum may be given in dosage of 0.5 to 1.0 c.c. intramuscularly, in the upper arm, with a tourniquet placed above the site of injection. Should a reaction occur, the tourniquet is tightened to stop rapid absorption of the injected serum, and an injection of 3 to 5 minims of adrenalin, 1-1000 solution, should be given into the other arm, and the dose repeated if necessary.

Typing of Sputum

The rapid-stained-sputum-slide method of Neufeld, as modified by Sabin, as noted earlier in the paper, should now be made. If no facilities are available for such typing, a specimen of sputum *coughed up from the lung* should be collected in a clean container, without preservative, and shipped to the State Board of Health for typing (be sure to include a brief history of the onset of the illness) and a telegraphic report awaited. While awaiting the typing report, if the onset suggests a frank lobar pneumonia, it is considered good practice to proceed with a preliminary injection of 10,000 to 20,000 units of a polyvalent serum Type I and II.

Administration of Serum and Dosage

The required dosage of serum is unknown and varies with each case. In general, it is known that the serum is most efficacious when given as soon after the onset of the disease as possible. Favorable results have been reported from the use of serum given up to ninety-six hours after the onset. After that time the serum has been proved to be less effective, although if toxemia is marked and the type of infection is known, serum may be tried, but the dose necessary is likely to be much larger. At present in Type I cases, a total of 60,000 units of antipneumococcic serum divided into three injections at two-hour-intervals is recommended.

The first dose is a test dose and but 2 c.c. of the serum should be injected *with extreme slowness at a rate of not faster than 1 c.c. per minute by the watch.* The serum should be warmed before injection by placing the vial in water at body temperature. Do not use cloudy serum.

Following the first dose an interval of two hours is allowed to elapse, and a second dose consisting of the remainder of the first vial is given also slowly *during a period of five to ten minutes.* After a further delay of two hours, a third dose totalling 20 c.c. or more of serum is

given *during a period of about fifteen minutes.*

For several hours after the first three doses, totalling about 30 c.c. or 60,000 units, have been given, no change is likely to be observed, but within eight to twenty-four hours after starting the first dose, there is usually marked improvement. This is more noticeable in patients with Type I infections than in those with Type II infections. The temperature falls, pulse rate becomes slower, toxemia lessens, the area of consolidation ceases to extend, and a previously positive blood culture usually becomes negative.

The most reliable guides to the need of further dosage are the level of the temperature and the presence or absence of bacteremia. If facilities are not available for blood culture, then the need for additional serum may be estimated from the clinical aspect. If additional dosage is deemed necessary, it may be given in amounts of 20,000 to 40,000 units at intervals of two to six hours. Under no circumstances should more than 50 c.c., or 50,000 units, be given at any one time. Treatment of Type VII, or VIII, may require a total of 60,000 to 120,000 units or more of serum.

Serum Reactions and Their Treatment

Two types of reactions may occur either at once or within a short time after the injection. These are the immediate or allergic, and the thermal or chill reaction. Certain symptoms are common to both types of reactions, and at times it is difficult to distinguish between them. The physician should remain at hand or be immediately available for from 30 to 45 minutes after the injection.

In a characteristic type of allergic reaction there may be flushing of the face, dyspnea, cyanosis, lumbar or abdominal pain, a rapid, weak, or irregular pulse, apprehension, and a feeling of sternal oppression, or various combinations of the above. In the Massachusetts Pneumonia Study, such symptoms were usually transient and of a mild or moderate intensity and were noted to have occurred within thirty minutes in 8 per cent of serum-treated cases. Treatment for such reactions consisted of subcutaneous, or intravenous injections of 0.5 to 1.0 c.c. of 1-1000 adrenalin.

In the second type the above reactions were sometimes combined with urticaria and an acute asthmatic attack. These reactions occurred in patients who gave a history of previous serum administration. Repeated doses of adrenalin should be given for these reactions.

The milder immediate reactions of an allergic type are not a contra-indication to further serum treatment, but subsequent individual doses should be smaller than usual. *The occurrence of urticaria or an asthmatic attack is a contra-indication to further injections.*

Thermal reactions occurred in 20 per cent of the Massachusetts Pneumonia Study. They usually were not serious and their treatment was symptomatic. *Adrenalin was of no value in the treatment of thermal reactions.* The usual serum therapy régime was not modified because of the milder thermal reactions. In cases with the severer type of thermal reaction, a time interval of three to four hours was allowed to elapse after the chill before a subsequent diminished dose of serum was administered. Attempts to produce chill-free sera have been partially successful.

It is desirable to warm the serum to body temperature, inject it slowly, and reject serums containing a precipitate as unfit for use.

Serum sickness occurs from four to twenty-one days after the serum treatment in about one-third of the cases treated. It is usually mild. Treatment consists of soothing applications to the skin, ephedrin by mouth, and salicylates for joint pains. There may be more than one bout of serum sickness.

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THE USE OF THE GASTROSCOPE*

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ALTHOUGH gastroscopy has been a recognized method of diagnosis in Europe for some years, it is a relatively new procedure in the United States. Until about four years ago very little gastroscopy had been done in this country. Since then a number of gastroscopists have come to the front. We began to use this method in 1933 and have performed 450 gastroscopies since that time.

In the course of this discussion comparisons will be made between gastroscopy and roentgenology. The x-ray has been the most accurate method of diagnosis in gastric diseases and there is no doubt that the x-ray method in the hands of a competent roentgenologist is the best single method of gastric diagnosis today. The roentgenologist may well boast of the great progress made in his field. Gastroscopy is not offered as a substitute for the x-ray method; it is rather offered as a supplement. Gastroscopists merely want to point out that this new method of direct visualization of the stomach wall offers additional information in those lesions already diagnosed by the roentgenologist. In those diseases not readily diagnosed by the x-ray method, however, gastroscopy offers valuable information which can be obtained only by this method. The roentgenologist makes his diagnosis on changes in form and mobility of the stomach wall as demonstrated by the shadow cast by a barium filled viscus. The gastroscopist, on the other hand, makes his diagnosis by actual observations of changes in form, color, consistency and mobility of the stomach wall. I am sure that all of us would agree that cystoscopy offers valuable information which cannot be obtained from a cystogram. I do not mean to convey the idea that I believe the cystogram to be unnecessary but no one will deny that much more knowledge concerning the actual condition of the bladder mucosa is obtained from a cystoscopy. I once asked Professor Friederick von Mueller what he thought of gastroscopy as a method of gastric diagnosis. He answered me in two ways. First he assured me that he had adopted this means of diagnosis in his clinic, which fact he said was enough evidence that he thought it to be a valuable adjunct. He also reminded me that when he was a young man the medical profession ridiculed cystoscopy and that he had lived to see the day when this procedure was a common and accepted one. He assured me that gastroscopy would also be an accepted method of diagnosis in years to come.

Before discussing our observations, I believe it might be well to describe the method to those among you who are not familiar with it. The gastroscope may be of several types. The open scope, which is a rigid tube much like an esophagoscope, allows direct vision of the field immediately ahead of the tube. It is the only instrument usable for the removal of foreign bodies. It is limited in its use because only a very small portion of the greater curvature can be brought into view. The other instruments use indirect vision. They are of two types: a rigid scope and a flexible scope. Each consists of a long tube containing an optical system with an eyepiece at the upper end and a prism near the lower end which allows a view at an angle of 90 degrees to the scope. An electric bulb is placed near this prism and a rubber tip is placed at the lower end. The stomach may be inflated with air by means of a bulb at the upper end. These instruments permit a very complete view of the gastric mucosa. The flexible type as a rule allows a more complete visualization of the lesser curvature and the pylorus while the rigid scope because of its interchangeable optical systems allows a better view of the cardia. The view obtained is very clear with either gastroscope. The mucosal folds, peristaltic waves, changes in color and form are easily perceived.

The method is not as disagreeable to the patient as esophagoscopy or bronchoscopy and many patients would rather be gastroscoped than be lavaged with an ordinary Ewald tube. Most physicians not familiar with the method suppose

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it to be very dangerous. If ordinary care is exercised in the selection of subjects and the manipulation of the instrument, the method is practically without danger. The flexible instrument is probably slightly more safe than the rigid tube. For example, Henning[1] reports a series of 1,200 gastroscopies with the rigid tube and 1,400 with the flexible tube without a fatality. When I first used this instrument I was not as careful in the selection of my patients as I am now. Consequently, when I attempted to gastroscope an elderly man, age 80, a necrosis in the posterior pharyngeal wall with a resultant fatal mediastinitis occurred. Since then, I have exercised greater precaution and have had no more fatalities. There is also the danger of perforation of the stomach either by actual contact with the instrument or by over-inflation with air. These accidents are very rare. The only difficulty of this type that I have encountered was a small tear in the gastric mucosa due to over-inflation with air. This lesion produced no symptoms. I believe that now we are justified in stating that there is practically no danger from gastroscopy in the hands of careful operators.

Before we are able to discuss the abnormal conditions in the stomach it is well that we fix in our minds the normal pictures of the gastric wall as obtained through the gastroscope. When the normal stomach is inflated we see that the mucosa is a smooth membrane with a pinkish-orange color. The rugæ appear as elevations with valleys of normal mucosa between them.

The rugæ can usually be wiped out with sufficient air pressure; in fact, if this is not possible an abnormal condition is present. One obtains a fairly complete view of the anterior and posterior walls and the greater and lesser curvatures. The angle in the lesser curvature appears as a crescent shaped ridge at the beginning of the antral canal. The antral canal can as a rule be well visualized although it is often difficult to visualize that small portion of the lesser curvature in the antrum. The pylorus can be easily seen as it opens and closes in tune with the peristaltic waves that roll down the antral canal. Peristaltic waves are only rarely seen in the body of the stomach. Changes in form, consistency, color or mobility of the mucosa and stomach wall indicate pathology.

The subject of gastritis is a very interesting one. In the older literature gastritis was a common topic of discussion. The older clinicians frequently diagnosed certain symptom-complexes as gastritis. However, with the advances made in pathology, that branch of medical science was eventually considered the last word in diagnosis. The pathologists began to notice that they never found the gastritis diagnosed by the clinician. It is true that they were able to recognize the severer forms of chemical gastritis but all other diagnoses of gastritis were taboo because the pathologist could not find the lesion at autopsy and of course only rarely did a patient with gastritis die of his disease. Even today it is true that the pathologist does not find these lesions in a routine autopsy for the reason that immediately following death the gastric ferments digest the mucosa. In fact it has been very difficult for the pathologist to describe the appearance of the normal mucosa from autopsy material. Since the advent of gastroscopy and the emphasis which has been placed on gastritis by this method of diagnosis, pathologists have been on the watch for biopsies of these lesions and to their credit it must be said that they have readily admitted that gastritis does occur, probably not infrequently.

It is in the field of gastritis that the gastroscope has made its greatest contribution up to the present time. Every clinician has seen patients with symptoms of epigastric pain immediately following or coming on one to two hours after meals, belching of gas, feeling of fullness in the epigastrium, nausea or vomiting. In these patients the roentgenologist usually finds nothing. The clinician has been prone to label such people neurotics and has as a result disregarded these symptoms. It is in just these patients with negative x-ray studies and gastric complaints that the gastroscopist can be of great help. It is not infrequent for him to find that these patients are really suffering from lesions in the stomach. Most commonly he will find some form of gastritis. By the term gastritis he does not necessarily mean to infer that these lesions are inflammatory in nature. It may well be that in the future we will find that such lesions are on some other than an inflammatory basis.

Schindler[2] has classified gastritis into three main types. The most common form he has

called hypertrophic gastritis. These lesions are commonly found in patients with gastric complaints such as pain after meals, belching of gas and a general feeling of fullness in the stomach. Gastroscopically this form of gastritis is easily recognized. The rugæ are larger than normal and can not be wiped out by air pressure. They seem to be made up of numerous small nodules of mucosa with fine narrow valleys between them. Viewed in profile the mucosa assumes a velvety appearance. Very frequently this form of gastritis is accompanied by single or multiple small erosions which may vary in size from one to five millimeters in diameter. These erosions appear as shallow punched-out areas in the mucosa and look very much like small ulcers or canker sores. This form of gastritis is very common in patients who have been thought to be suffering from spastic colon or irritable bowel. In my own mind, I am not yet convinced that it may not be on a neurogenic basis. It is entirely possible that future study will reveal that it is not an inflammatory reaction at all. These patients do not seem to react well to any form of treatment. Despite rigorous ulcer or bland diet regime these lesions, although they may improve temporarily, do not seem to be permanently cured. The erosions, however, will disappear on rigorous treatment and will usually remain healed as long as the patient will remain on such treatment. We have had many examples of this type of gastritis in the last four years. Just recently, I saw a patient who for six years had been having epigastric pain coming on two hours after meals relieved by diet and in whom x-ray has been unable to reveal any signs of ulcer. Gastroscopy revealed a localized hypertrophic gastritis rather high on the anterior wall with several small ulcerations ranging in size from two to five millimeters in diameter. He has been on an ulcer diet for two weeks but as yet he has not had complete relief from symptoms.

Atrophic gastritis is a frequent accompaniment of pernicious anemia although it may occur in the absence of this disease. Its recognition is very simple. Whether it is a result of an old hypertrophic gastritis or is a primary form in its own right is a debatable question. For the present we shall leave this as an open question.

That form of gastritis known as chronic mucosal catarrh has been a very interesting entity. It consists of a superficial inflammation of the mucosa with marked discoloration of the mucosa and a dirty necrotic greyish exudate lying between the folds. It is commonly found in carcinoma of the pylorus with obstruction, passive congestion, and in some cases of gastro-enterostomy. We have found it to be present in the majority of those with gastric complaints following gastro-enterostomy. The symptoms of this lesion simulate those of ulcer and very frequently an erroneous clinical diagnosis of gastro-jejunal ulcer has been made in such cases. This lesion improves markedly under a strict ulcer regime but is prone to recur with the slightest dietary indiscretion. I believe that the occurrence of this form of gastritis is one of the most serious objections to the use of gastro-enterostomy as a treatment for duodenal ulcer. Although it might be avoided by correctly planned gastro-enterostomy openings, it seems that even in the hands of excellent surgeons the operation is not infrequently followed by this lesion. In contradistinction to this procedure, gastric resection seems to produce much better results. In our series of cases, I have never seen chronic mucosal catarrh or any other form of gastritis in a resected stomach. I believe that this constitutes a strong argument for the use of gastric resections rather than gastro-enterostomy in the surgical treatment of obstructive duodenal ulcer. It may be that at some future date we will find this form of gastritis in a resected stomach but the indications at present are that it will be a rare condition.

Gastric ulcers are easily recognized. These lesions appear as round or oval punched out areas with white necrotic centers surrounded by reddened mucosa. This mucosa is not raised but remains on the same level as that around it and the rugæ radiate out from the margin. As healing occurs the ulcer fills in from below and from the periphery. The white necrotic center sloughs out and is replaced by granulation tissue. The edges become red and encroach upon the ulcerated area. When the ulcerated area becomes very small it is still surrounded by a ring of reddened mucosa. After the ulcerated area is healed, the mucosa in the area of the original ulcer may remain reddened for quite a long period of time. Should the healing process be in-

terrupted the ulcer breaks down and the necrotic center again sloughs out. Gastric ulcer is, as a rule, easily diagnosed by the x-ray method. Nevertheless, we have been able to discover small ulcerations which were not picked up by the roentgenologist. It is obvious that small lesions 1-2 mm. in diameter could be easily missed if they should occur just off the lesser curvature on the anterior or posterior walls. We have also been able to demonstrate that a lesion which simulated a gastric ulcer on x-ray was in reality an outpouching of the stomach made up of entirely normal mucosa. Here again the x-ray silhouette could not be expected to differentiate between an ulcer with a deeply sloughed out center and a small outpouching of the stomach wall lined with normal mucosa.

Other inflammatory lesions of the stomach producing a scarring in the wall are frequently seen through the gastroscope. At times the mucosa over these areas is itself replaced by white scar tissue and on other occasions the mucosa is normal but the wall is stiff and distorted by scar tissue. Such lesions are often seen in the antrum associated with old duodenal ulcers which have produced considerable scarring in the adjacent tissues. At times the differential diagnosis between a gastric malignancy and such an inflammatory lesion is difficult on x-ray examination but the gastroscopist will as a rule be able to make the correct diagnosis.

Carcinoma of the stomach is usually correctly diagnosed by x-ray methods. Gastroscopically these lesions present a varied appearance. The lesions may vary in color from white to grey, blue or purple. They are raised above the normal level of the mucosa. The rugæ run up to this raised margin, which is as a rule markedly discolored. The ulcerations within the lesion are usually irregular in outline and as a rule show numerous bleeding points. Because of the difficulty in the x-ray examination of certain portions of the stomach it is occasionally possible to discover lesions by gastroscopy which were not found on x-ray examination. In one such case we were able to discover a lesion approximately 4 cm. in diameter which repeated x-ray examinations were unable to locate. The patient was explored and the lesion was found to be present exactly as described on gastroscopy. Gastric resection was performed and the patient is alive and well at this time, sixteen months after operation. Although such cases are rare, an example such as this case illustrates the importance of gastroscopy when gastric lesions are suspected and a negative x-ray examination is obtained.

The patient with gastric hemorrhage presents an interesting medical problem. Although the most common sources of bleeding in the stomach are carcinoma and ulcer, we all have been confronted with cases which present negative x-ray studies in the face of serious gastric hemorrhage. Even exploratory laparotomy frequently fails to reveal the source of bleeding. With the aid of gastroscopy we have been able to find that a large number of such cases bleed from rather small gastric lesions. The most frequent source of such hemorrhages is a small erosion which may be pinpoint in size. Such erosions may be single or multiple. I have on numerous occasions seen a fairly copious stream of blood pouring from such a lesion. The presence of varices in the stomach and not in the esophagus constitutes another source of bleeding. We have seen four such cases in our series. At times hypertrophic gastritis bleeds very easily on the slightest pressure with the gastroscope. It is entirely probable that such lesions could easily bleed when food particles come into contact with them. I believe that in all cases of gastric hemorrhage for which no cause can be found, gastroscopy should be done as this method alone enables us to discover the source of bleeding. I do not feel that the presence of bleeding is a contraindication to gastroscopy.

Conclusions

Gastroscopy is a new and safe method of examining the stomach. It offers valuable additional information which can only be obtained by this method. In the field of gastritis it is the one reliable means of diagnosis at the present time.

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THE EXTERNAL USE OF ALOES

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IN 1935, Collins reported treating roentgen burns with the leaf of Aloë vera. About that time, I was interested in a case of palmar eczema. The lesions had been of long duration, had resisted every type of treatment and were painful and annoying. I obtained enough of the leaf for four applications, which I made on four successive nights. The leaves were split, the gelatinous surface applied to the palms and the material retained with bandages. At the end of the four treatments the lesions were healed and the patient had had no further trouble up to the time of the writing of this communication on December 20, 1936.

Further Clinical Experiences

Ointments are not generally used as surgical dressings but, in my experience, the results obtained and the ease with which the aloes dressings were removed, without adhering or causing pain, seem to be points in favor of their use. As will appear, I used not only an ointment of aloes, but also powdered aloes. These preparations were applied in the routine of a general practice and on empiric grounds only. The reports of results will be faithfully, if briefly, recorded.

Ulcers on Amputation Stumps.—I received a supply of the leaves from the Island of Aruba in the Dutch West Indies, and these I used in the treatment of several ulcers which had appeared, after amputation, on the stumps of the legs of a single individual. Because lymphedema had developed and the condition that was present resembled elephantiasis, amputation had been performed a second time, together with removal of some of the veins. The circulation in the stumps was poor, and in 1932 several large ulcers had developed. These had shown no inclination to heal under various kinds of treatment. On the left stump were three ulcers, one of which measured 5 by 13 cm.; the other two were about 3 cm. in diameter. On the right stump was a single, deep ulcer, also about 3 cm. in diameter. Within twenty-four hours after application of the leaf of Aloë vera, pain had practically disappeared and the edema was much reduced. The smaller ulcers healed in about two weeks, leaving practically no scar. The large ulcer made good progress but my supply of leaves became exhausted.

I had become much interested in the drug, and finding that the ordinary commercial powdered aloes was prepared mostly by natives, who dried the pulp of the leaves in the sun, I thought this sun-dried product might retain the properties of the fresh leaf. I obtained a supply of Socotrine aloes, the best grade of powdered aloes. This I made into an ointment with a lanolin base and applied it to the large ulcer. The relief from pain and healing continued about as it had done when the fresh leaf had been applied. However, after several months there appeared to be little progress. I then discontinued this treatment and used hot packs, scarlet red ointment, balsam of Peru, calamine ointment and zinc oxide ointment, one after another. Healing not only stopped but the size of the ulcer seemed to be increasing. I returned to the aloes ointment and also applied powdered aloes; the latter seemed to be a little more effective than the ointment. At the present time* the large ulcer remains unhealed, and in the past few months has made little progress. It is now 5 cm. long and 3 cm. wide. Because of the bad circulatory condition, surgical treatment seems inadvisable.

Pruritus Vulvæ.—Seven cases of pruritus vulvæ have been treated and in all but two the results have been satisfactory and prompt. Of the two cases in which the results were less satisfactory, in one the condition was of long standing and an obstinate vaginal discharge was present. Also, in this case there was persistent perspiration of the groins; the perspiration appeared to be irritating and was difficult to control. This patient, although much improved after two months, is still under treatment. She has had much relief from the itching and the skin has a much better appearance than before. Nevertheless, the irritating vaginal discharge is refractory. In the other of these two cases progress has been

*Note.—August 11, 1937. The large ulcer has been completely healed for two months.

slow, but the condition has improved and the patient is still under treatment.

Of the cases of pruritus vulvæ, the following one was particularly encouraging. The patient was sixty-four years of age. The condition was very severe and the skin about the vulva and down the inner aspects of the thighs was thickened and purplish. Considerable treatment had been ineffective. The woman was in a highly nervous state and was unable to sleep. When treatment was begun with the aloes ointment, administration of sedatives was continued. The intense itching and burning was almost immediately lessened; in two weeks the skin had become practically normal in appearance and the irritation had been completely relieved.

Ulcers of Advanced Mammary Carcinoma.—A woman, eighty-six years of age, had a carcinoma of the left breast of one year's duration. Because of her age and for other reasons, the condition was considered inoperable. In March, 1936, a crater-like ulcer developed and became a little more than 3 cm. in diameter. The discharge was profuse and very foul-smelling. After various applications had been tried without effect, aloes ointment was applied. In a few days both the discharge and the odor were controlled. Odor could not be detected more than a few centimeters away from the lesion after removal of the dressing. The application was made on gauze and this was easily applied and easily removed twice daily. Granulation developed promptly and the crater-like ulcer became level with the surrounding tissue. This treatment was continued from the latter part of March until the patient died, December 11, of abdominal metastasis. During this time the tumor became larger and more nodular, but after the depression filled there was little change in the size or appearance of the ulcer.

Ivy Poisoning.—A pregnant woman twenty-two years of age had small lesions on the wrists caused by poison ivy. However, almost the entire inner aspects of both thighs were equally involved as to area and degree. The irritation in these areas was violent and inflammation and blebs caused much distress. A solution of potassium permanganate was applied to the right thigh and the woman was given some of the solution with which to continue treatment at home. To the left thigh aloes ointment was applied liberally and the area was covered with gauze. The patient lived in the country, but she returned as directed on the second day. The lesions on the thigh that had been treated with potassium permanganate had somewhat dried but were still inflamed and very uncomfortable. The left thigh, to which aloes ointment had been applied only once, had caused but little discomfort and the skin was normal in appearance, with the exception of a little remaining moisture and redness where the blebs had been. The patient was given ointment for the right leg also and was not seen again until her confinement two weeks later. She stated that the condition of both thighs had cleared up promptly.

Burns.—A man, aged twenty-six years, had stepped into a pit containing boiling water emptied from a cooker in a canning factory. The injured foot and leg were scalded to within 8 cm. of the patella. Most of the outer layers of skin came off when the stocking was removed. Over the malleoli, burns extended through the entire integument. In a first aid station a proprietary ointment had been applied. In an effort to remove the ointment and dead skin, I applied, at first, warm, moist packs of solution of boric acid. On the following day, and thereafter, liberal applications of aloes ointment were made. The patient had little pain and the dressings were easily removed. There was no evidence of infection. In ten days there were no raw areas, although the skin looked thin, red and shiny. The patient was dismissed and returned to work on the nineteenth day after the accident.

In another case, in which a large area had been scalded, and in several cases of moderate burn, treatment and results were similar.

Other Conditions.—In addition to the cases cited, the ointment or powder was used in place of ordinary antiseptic substances in treatment of carbuncle, small infections and abrasions. In one case of "winter itch" also, a very dilute alcoholic solution was employed.

Unfavorable Effects in Three Cases.—In one case of ulcer of the leg in which aloes ointment was employed catharsis was present for one day. This may have been attributable to the aloes. Because of possible absorption, therefore, aloes probably should not be used on mucous surfaces, such as those of the vagina, except with caution.

Moreover, in two cases of psoriasis, there developed what appeared to be allergic erythema bordering the original patches. With the exception of these cases, I have not seen any untoward results from the use of the ointment or the powder.

Ancient and Modern Uses of Aloes

The drug has maintained a place in the history of medicine since the time of King Tutankhamen, 4,000 years ago. It was known to the ancient Egyptians, is mentioned in Arabian medicine, and was employed down through biblical times and the dark ages to modern times. Aloes is used but little at present in the United States, except by veterinarians; nevertheless in the Twenty-second United States Dispensatory six pages are devoted to it. In former times aloes was said to be useful in amenorrhea and as an abortifacient. It has long been used as a stimulant to the lower bowel in constipation and as a general tonic. It is mentioned as being useful in the treatment of abrasions, fissures, and so forth, and is a component in the official compound tincture of benzoin. Aloes was formerly used as an embalming agent and perhaps was one of the substances used by the ancient Egyptians in their now lost art of embalming. In the Bible, John 19, verses 39 and 40, is the following passage: "And there came also Nicodemus, and brought a mixture of myrrh and aloes, about an hundred pounds. Then they took the body of Jesus and wound it in linen clothes and the spices, as the manner of the Jews is to bury."

The fresh leaf of the plant is still used by the natives of various countries. The Seminole Indians in the Everglades of Florida use it for treatment of wounds and burns. A friend from South Africa reported to me that the natives there used the leaf for treatment of sores and wounds. Another friend has told me that he has often seen it used by the peasants in southern Italy for treatment of ulcers, wounds, and so forth, and that he himself has used it for severe sunburn with great relief.

The Plant and Its Preparations

The genus Aloë embraces about a hundred species, which grow from a few centimeters in height to plants 6 meters high. The leaves of Aloë vera somewhat resemble the leaves of the century plant. They are about 40 cm. long and are dark green; sometimes they are mottled with brownish spots. The leaves are flat on the upper surface and convex on the under surface and the margins are armed with reddish thorns. The base of the leaf is about 1.5 cm. thick. The integument is thin and fibrous and the interior of the leaf is filled with a gelatinous substance resembling lemon gelatin.

The plant grows widely in warm countries. It is found wild and is cultivated in countries bordering the Mediterranean Sea and in India, Africa, China, and the Islands of the Indian Ocean. It is cultivated in many of the islands of the West Indies, especially in Barbados, Curaçao and Aruba, and it is found in Mexico and Florida. I have not seen mention of its cultivation in any of the Pacific regions.

The various commercial products are named principally from the localities where they are produced. Socotrine aloes, made principally from the species Aloë perryi, but also from Aloë vera, comes from the Island of Socotra in the Indian ocean. Barbados and Curaçao aloes are produced mostly from the species Aloë vera, which is grown on various islands in the West Indies. There are many other kinds of aloes which are known by the following descriptive adjectives: "Cape," "Natal," "Zanzibar," "Aganda" and "Crown." Besides the preparation from Aloë vera and Aloë perryi, a number of preparations, mostly inferior, are made from other species and are official in the United States Pharmacopeia.

Because commercial aloes, it is said, is prepared principally by natives, who remove the jelly-like interior of the leaves and dry it in troughs or other containers in the sun, it often contains much foreign matter. For this reason, in the United States Pharmacopeia it is advised that a purified aloes be made by dissolving the powder in alcohol and then straining and drying the filtrate. I have used the crude powder, as I said before, because I thought some of the properties might be impaired by the alcoholic treatment.

Owing to the fact that there are so many varieties of commercial aloes, it seems possible that the results may not always be uniform. The material I have used has been the best grade of Socotrine aloes, and the results have been fairly uniform. The ointment which I made contains 1 drachm (4 gm.) of powdered aloes and 1 drachm of calamine to the ounce (30 gm.) of

white petrolatum. The calamine forms a sort of paste and makes the ointment more adherent. The dusting powder which I also have said that I used in some cases, has not caused any irritation or other undesirable effects.

Summary

The fresh leaves and the aloes ointment and the powder appear to have the following properties: 1. They relieve pain, burning and itching. 2. They have some sort of antiseptic action. Infected lesions quickly become clean and exude little or no pus. 3. They seem to stimulate rapid granulation and formation of new tissue so that denuded areas appear to heal more rapidly than with other agents. They are effective in eliminating the foul odors that accompany infection of broken down carcinomas, ulcers and so forth.

CASE REPORT

A CASE OF MALARIA DEVELOPING IN MINNESOTA*

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ALTHOUGH malaria remains endemic in our southern states, and in the past has occurred frequently in the states on the Mississippi River and its tributaries, it seldom has been encountered in Minnesota in recent decades except among transients from areas where the disease is endemic or among patients with paresis who have been artificially inoculated for therapeutic purposes. However, with modern methods of rapid transportation, such as the automobile and aeroplane, the possibility of transportation of infected mosquitoes must be considered from a public health standpoint. Furthermore, it is known that anopheline mosquitoes exist in Minnesota. Because of these facts, it has been thought advisable to record the following case of malaria developing in a native Minnesotan.

A man, aged twenty-one years, came to The Mayo Clinic on June 2, 1937, from his rural home at Minneiska, Minnesota. He had always lived in Minnesota and never had been out of the state farther than Alma, Wisconsin. He had had pneumonia in 1933 and had been operated on for acute appendicitis in 1935. Several months before he came to the clinic, the cervical lymph nodes on the right side had enlarged somewhat but this enlargement had subsided. Since December, 1936, he had had three or four mild chills, profuse perspiration, malaise, and slight fever, occurring in the evening. The following morning he had felt normal except for some weakness, which had persisted for two or three days. About four weeks before his admission at the clinic he had had almost daily chills, lasting from twenty minutes to two hours, followed by drenching sweats. These had been associated with severe aching of muscles and bones, headache, and fever as high as 104° F. General malaise had been marked. The patient's weight had decreased from 147 to 124 pounds (67 to 56 kg.) and he had lost much in strength. He had been in a hospital near his home for one week where, he stated, a tentative diagnosis of malaria had been made and he had been given some medicine. He then had been at home for two weeks, during the first of which he had had a mild chill every second day, followed by slight fever. Because of continuation of these symptoms, he stated that his physician then had questioned the diagnosis of malaria.

The day after the man came to the clinic he had a chill, followed by a temperature which he said was 103° F. He was not under observation at the time when he had this temperature. Physical examination gave evidence of moderate loss of weight; the right cervical lymph nodes were palpable and the spleen was readily palpable. During the period he was under observation the highest temperature reading was 99.2° F. Various laboratory examinations gave normal or nearly normal results, until, on June 10, study of thick smears of the blood disclosed the presence of a plasmodia. On a subsequent smear, typical ring forms of Plasmodium vivax were identified, although they were very few. Atabrin, one tablet, three times a day, was administered for five days, and the patient was instructed to take a second course of the same treatment in from one to two weeks.

*From the Division of Medicine, The Mayo Clinic, Rochester, Minnesota.

EDITORIAL

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BUSINESS MANAGER
J. R. BRUCE

Volume 20 OCTOBER, 1937 Number 10

Dr. George H. Simmons

WITH the death on the first of September of Dr. George H. Simmons, former general manager of the American Medical Association and editor of *The Journal*, came the end of a long and influential career, closely associated and largely responsible for the remarkable growth in influence of our national medical organization. His crusader-like and vigorous attack on fraud which was manifest in the nineties when he practiced in Lincoln, Nebraska, and which led to the recognition by the Chamber of Commerce of that city of the debt owed him, was continued during his association with our national organization. His extraordinary literary ability which he used at an early age in obtaining his education was devoted to the national journal during his editorship.

After careful deliberation the Board of Trustees chose Dr. Simmons to be General Secretary and Editor in 1899. When he retired in 1924 he had been editor for twenty-five eventful years of the Association's existence. It was he who instigated the reorganization of the Association and the adoption in 1901 at the meeting of the Association in Saint Paul of a new and more representative constitution. His regime saw the formation of the Council on Medical Education and Hospitals, the Council on Pharmacy and Chemistry, the beginning of the publication of the *Quarterly Index*, the *American Medical Directory*, and the various *Archives* to supplement *The Journal*.

It is not surprising that, in his vigorous and continuous attack on fraud, he became the butt of much abuse. Doubtless his refusal to reply to unjust criticism gave the impression in some quarters that "silence gives consent." The record of progress made in medical organization, education and publication during his incumbency and the part he played in instituting many new activities and reforms speak for themselves.

Poliomyelitis in Minnesota

ACCORDING to the latest reports of the poliomyelitis situation there has been a widespread increase in incidence this year, especially in California, Texas, Oklahoma, Louisiana, Mississippi, Alabama, Arkansas, Tennessee and North Carolina. At the same time a considerable number of cases have been reported in Massachusetts, New York, Ohio, Indiana, Illinois, Michigan, Kansas and Nebraska.

Each year a certain number of cases is reported in Minnesota. This year three cases were reported in January, one in February, one in April and one in June. From July 1 to September 11 a total of 159 cases was reported, thirteen with first symptoms in July, fifty-six in August and ninety up to September 11, only one of these outside of Minneapolis, Saint Paul and Duluth. A comparison of the number of cases so far this year with the total number in Minnesota in recent years is interesting. In 1925 there were 955 cases with 145 deaths; in

1930, 479 cases with thirty-seven deaths; in 1931, 811 cases with sixty-six deaths; in 1932, 124 cases with ten deaths; in 1933, 383 cases with thirty-seven deaths; in 1934, 113 cases with twenty-one deaths; in 1935, 99 cases with ten deaths.

When all the evidence is summed up, the profession still has little to offer in the prevention or cure of poliomyelitis. Its occurrence is not restricted to limited areas, and often a large number are isolated cases. The peak of an epidemic is generally reached about the middle of September, the exact time following the opening of the schools when one would expect the epidemic to increase rather than diminish, showing that poliomyelitis does not behave like most contagious diseases.

Last month we referred editorially to Dr. Armstrong's report of the failure of nasal spraying with picric acid and alum in preventing infection. Recently Dr. Armstrong has called the attention of the Minnesota State Board of Health to the fact that, while nasal sprays are very effective in preventing infection in animals, application of any medication with an ordinary atomizer fails to reach the required area. Even Dr. Peet's method of inserting an atomizer tip above the middle turbinate is not practicable in young children.

The use of zinc sulphate solution as a nasal spray, too, is entirely experimental. Reactions are frequently so severe that the carrying out of the prescribed two successive daily treatments and repetition at intervals of two weeks is likely not to be consummated. The Minnesota State Board of Health takes the stand, and it seems to us rightly, that as long as the experimental studies on human beings have not proved the value of nasal sprays in the prevention of poliomyelitis and since there is some danger associated with their indiscriminate use, the Board does not recommend them.

In regard to active treatment of poliomyelitis there has been much difference of opinion. Our State Board of Health has never been very enthusiastic about the value of convalescent sreum although it has made it available in recent years. As a result of the work of Dr. Park in coöperation with the New York City Academy of Medicine, reported in 1932, and again in more detail by Fischer, and also that of Kramer. Aycock, Solomon and Thenebe, the State Board of Health has gradually withdrawn from this activity and this year has not procured or distributed convalescent poliomyelitis serum. A critical evaluation of the results of serum therapy by Harmon which appeared in the *American Journal of Diseases of Children* in 1934 and a report by Rosenholz at our State Association meeting in 1933 both failed to demonstrate that the serum has any value in the preparalytic stage. It has been given largely in the hope it would be of benefit.

Oculists and Optometrists

THE medical profession and particularly oculists have recognized for some years the part that ignorance on the part of the public as to the difference between an oculist and an optometrist plays. It is safe to say that many people do not know that many optometrists who call themselves doctors on the basis of their D. O. diplomas are not doctors of medicine. They do not know that the majority of optometrists are not competent to detect diseases of the eye from which a large percentage of their patrons are suffering.

Inasmuch as the medical profession in general and oculists in particular are open to the accusation of bias when they attempt to publicize the present situation, the article entitled "Optometry on Trial" by Roger William Riis which appeared in the August number of the *Readers' Digest*, written not only by an M.D. but by one presumably unprejudiced, should therefore be of some value. Certainly it produced a reaction on the part of optometrists and oculists as presented in the September issue of the same magazine.

The article is based, not on preconceived ideas, but on the results of an investigation in which some six individuals consulted optometrists from Boston to the Rockies and in which some $1,500 worth of glasses were purchased. The results are rather astounding. A girl with normal vision only on one occasion was told she needed no glasses. One individual suffering from glaucoma, iritis and squint was not once correctly diagnosed, and less than half the glasses prescribed were ground according to prescription.

According to the article there are 22,000 optometrists in the country and they sell $75,000,000 worth of glasses a year. It was found that

88 per cent of those investigated offer free examinations and thus have to furnish glasses to make any profit. It is no wonder that to the commercial element among the optometrists the temptation to prescribe unnecessarily or to neglect to refer to an oculist those whom glasses do not correct is often too great.

The question arises, is optometry a profession or a business? The fact that most optometrists advertise and use all possible means to promote sales indicates they are for the most part business and not professional men. If optometry is a profession it is necessary for their organization to clean house.

It is true that the average physician knows little or nothing about refracting eyes, but is content to refer such patients to oculists. It is also true that optometrists know little or nothing about eye diseases, and, unless they can recognize glaucoma, iritis, cataract and optic atrophy, a distinct danger to the public exists in the situation.

The more publicity on the subject, the better for the public. Then when an individual's attempt to economize by saving the oculist's fee results in no improvement of vision, he or she will suspect some eye disease and consult an oculist.

In Memoriam

John J. Gelz
1883-1937

DR. JOHN J. GELZ, well known eye, ear, nose and throat specialist of St. Cloud, died June 26, 1937, following two years of retirement from active practice.

Born in St. Joseph, Minnesota, March 3, 1883, the son of John Francis and Carolina Haarman Gelz, who came to Minnesota with their parents at the time of the Civil War, Dr. Gelz was left an orphan by the death of his father in 1887, and he and his three sisters were reared by his mother.

Dr. Gelz attended school in St. Joseph and later the East Side High school in Minneapolis, where he graduated in 1904. He attended Hamline University and received his medical degree from the University of Minnesota in 1909. His internship was served at St. Joseph's Hospital in Saint Paul, and at St. Mary's in Minneapolis.

Beginning practice in Buffalo, Minnesota, Dr. Gelz moved after two years to Richmond, where he engaged in general practice for ten years. In 1921 he studied in Vienna and London and also took four months additional training in New York to prepare himself for eye, ear, nose and throat work. Following this he located in 1923 at St. Cloud. In 1927 Dr. W. T. Wenner became associated with him and in 1936 Dr. J. B. Gaida joined them.

Dr. Gelz was a Fellow of the American College of Surgeons, the Minnesota Academy of Ophthalmology and Otolaryngology, the Stearns-Benton County and the Minnesota State and American Medical Associations. He was also an honorary member of the St. Cloud Rotary Club, of which he was a past president. He was at one time president of the Stearns-Benton County Medical Society, and also of the St. Cloud Hospital staff.

Dr. Gelz married Catherine Wenner of Richmond on June 20, 1916. He is survived by his widow and three sisters.

Alexander Ridgway
1855-1937

DR. ALEXANDER RIDGWAY died at his home in South Haven, Minnesota, April 3, 1937, at the age of eighty-one years.

He was born in Columbus, Wisconsin, July 28, 1855. As a young man he left Columbus and went to Minneapolis, where he studied medicine at the Medical Department of Hamline University, from which he graduated in 1894. He located at Belgrade, Minnesota, where he practiced until 1929, when he moved to South Haven and continued the practice of medicine there until his last illness.

July 8, 1896, he was united in marriage to Miss Lena Hanson of Minneapolis. Two children were born to them, Russel of Minneapolis and Mable of South Haven. Dr. Alex was a member of a family of well known physicians, two of whom, Dr. Joseph Ridgway of Minneapolis and Dr. Alfred M. Ridgway of Annandale, survive. Dr. Alex Ridgway was a member of the State Medical Society and the Stearns-Benton County Society, and of the Masonic Woodmen, and Royal Neighbor lodges. He served several terms as Mayor of South Haven.

Dr. Ridgway was a man of exemplary habits and fine character and will be greatly missed in the community which he served and by his professional brethren who best knew him. He was an example of the rapidly thinning ranks of those pioneer country doctors who were forced to contend with the hardships of medical practice in rural districts in the olden days when the automobile and good roads were unknown, one who devoted his life to the service of the sick and afflicted of his community and of whom it may well be said that during his long professional career "He truly built many fires in cold rooms."

MEDICAL ECONOMICS

Edited by the Committee on Medical Economics
of the
Minnesota State Medical Association

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They Did Not Pass

Compulsory Sickness Insurance legislation was introduced in several state legislatures, including New York, Pennsylvania, and Wisconsin, during the last session. They failed in each.

It is rumored in informed quarters that Senator Robert F. Wagner of the state of New York plans to introduce a bill for compulsory sickness insurance in Congress at the next session. The rumor gains color by the fact that the Senator's son, Robert F. Wagner, Junior, introduced such a bill into the New York State Legislature and failed to secure passage of it at the last sessions.

It is also reliably rumored that the President himself is indifferent if not actually opposed to any move for socialization of medicine at present.

Tax Status

The tax status of state medical associations under the Social Security Act and under the income tax provisions of the Federal Revenue Acts is not made any clearer by decisions already handed down in Washington. According to a reliable report, four large state associations have been declared liable and four others, large too, and apparently engaged in the same general type of program, have been declared exempt.

Minnesota's status has not yet been determined.

Note on Osteopaths

It is rumored that an attempt will be made at the next session of Congress to give full rights to practice under the United States Employes Compensation Division regulations to osteopaths.

Osteopaths in Kansas are bringing action on appeal before the courts to determine whether or not they have the right to be registered under the Harrison Narcotic Act.

In that connection it is interesting to note, also, that Delaware and South Carolina have granted separate Basic Science Boards for Chiropractic.

No Pedicurists

This question is being asked: What is a chiropodist?

The chiropodists themselves want an official definition. They emphatically do not want a chiropodist to be synonymous with a pedicurist. It is their contention that theirs is a medical specialty which licensed physicians could but certainly do not practice. In Minnesota chiropodists are licensed to practice under the law.

Hypnotics

Laws forbidding the sale of hypnotic drugs except upon the prescription of a physician have been passed in Rhode Island and South Carolina. In Maryland the law has been amended to instruct physicians specifically to write prescriptions in calling for these drugs.

In Texas, on the other hand, a law limiting the sale of hypnotics has been repealed.

Ante-Nuptials

Of interest to physicians, also, is the fact that ante-nuptial examinations for syphilis and gonorrhea are now required by law in Illinois and Michigan. In New Hampshire a new law requires examinations for syphilis alone. In Oregon a law requiring examinations of bridegrooms, alone, has been amended to apply to both partners.

Thousands for Cancer

The National Cancer Institute was created by the last Congress with an appropriation of $750,000. Medical observers are hoping that this money may never be used to line pork barrels.

The plan of the Surgeon General to distribute it among already established institutions of research has their hearty approval.

New Quirk in Food and Drugs

S1077 is the number of the Wheeler-Lea bill, latest chapter in the long and, so far, futile effort to secure passage of new and up to date food and drug legislation. The latest bill which is apparently to be pushed at the next session calls for control of all advertising other than labelling for drugs and therapeutic devices.

The new quirk in this bill lies in the fact that it proposes to take enforcement away from the Department of Agriculture and give it to the Federal Trade Commission. The reason for this change is not clear, inasmuch as the Department of Agriculture is obviously equipped in experience and facilities for the task, while the trade commission is not. The possibility that such a change might open the way to undesirable political manipulation should not be overlooked.

Department of Public Welfare

A department of Public Welfare is still considered imminent in Washington. It is altogether likely, according to report, that the plan for reorganization of Federal offices will call for such a department. The gossip is that Harry Hopkins will be the department head.

In the meantime physicians have not changed their opinion that health activities of the federal government should be independent of social welfare and a qualified physician should be in charge.

Too Bad, Mr. Senator!

In June, Senator James Hamilton Lewis of Illinois rushed to Atlantic City to seek the advice, so he said, of the House of Delegates of the American Medical Association.

He was afflicted on behalf of his professional brethren in medicine.

He detected a new attitude in Washington and all over the country. The doctors would be obliged to accept a new interpretation of their position and responsibilities. He wanted to warn them.

Were they prepared to cope with this new interpretation: to protect scientific standards and fees in the face of it?

The Senator spoke as one upon whom an unpleasant duty had been forced against his will. He was the reluctant bearer of tidings.

True to his forebodings, a resolution embodying his worst fears was introduced in July into the Senate. In it the doctors were definitely ranged as civil servants, responsible to the government rather than to their patients, for the well being of the citizens. A system of federal medicine for the indigent and the low income groups was proposed with fees to be paid to the doctors by the Social Security Board.

The Senate was preoccupied with the Court bill and other matters, and took no action on the resolution.

The interesting thing to the doctors was that Senator Lewis, himself, introduced the distressful resolution.

What pain and embarrassment it must have caused the distinguished statesman! Remembering his sensitiveness as it was exhibited on the platform at Atlantic City, the doctors will have every sympathy for the violence which introduction of this resolution must have done to his finer professional feelings.

Sickness Insurance - Washington, D. C.

A new experiment in sickness insurance started in a strategic quarter last month.

It may or may not be—as some people believe—the first move in a quiet, administration-supported move to advance a federal program of sickness insurance as part of the general plan for Social Security.

This new experiment was introduced in Washington, D. C., for the 2,000 employes of the Federal Home Loan Bank Board and other agencies such as the Home Owners' Loan Corporation which come under the direction of the Board.

Clinic

According to a recent issue of the *United States News* a fully equipped clinic is being established in Washington under the direction of Dr. Henry R. Brown, Chief of the Tuberculosis Division of the Veterans' Administration. A staff of about 15 is to be hired for the clinic, including six or seven physicians.

Men with families will receive medical service for themselves and their families for a payment

of $3.30 a month. Single persons will receive the same benefits by paying $2.20 a month.

Members Have Choice

The service will include complete physical examinations and necessary medical and surgical care with certain important exceptions, together with hospitalization in a semi-private room for three weeks during a single illness, nursing and ambulance services.

Members of the association who do not care to patronize the clinic doctors may have the physicians of their own choice.

The exceptions noted include surgery of the brain and nervous system and any additional treatment after the medical director recommends confinement in an institution for mental, tuberculous, drug or alcohol addiction cases. Members are to be required, also, to pay for medicines, drugs, surgical appliances and orthopedic devices, eyeglasses and hearing aids. They are to pay, themselves, for radium and deep x-ray treatments; dental work; oxygen tanks or tents or materials; blood transfusions and special nursing service, unless the latter is ordered by the director.

"To Encourage Prevention"

The usual objectives and justifications are offered: to encourage prevention of illness by making prompt medical care available at all times without extra charge: to reduce costs to beneficiaries.

The sponsors for the plan hold out a blithe hope that illness in the group will be cut by one-half.

Experience with group sickness insurance does not always bear out this sanguine hope.

Doctors Should Watch

That the whole project deserves close watching by physicians goes without saying. If it is popular, extensions to other government departments are almost inevitable. Government services outside of Washington will undoubtedly find assistance among other facilities of the Veterans' Administration for the development of the service. The possibilities are enormous.

The responsibility of the medical profession now lies in careful scrutiny of the quality of service available and of the costs in proportion to the amount. If the plan shows the weaknesses that such plans have shown elsewhere the facts should be pointed out promptly and decisively and before the project grows to unmanageable proportions.

Before the Court

Monthly Editorial Prepared by the Medical Advisory Committee

A speaker at a recent medical meeting emphasized certain points in the appearance and conduct of a doctor in court cases which your Medical Advisory Committee believes should be stressed to the membership of our Association.

1. *Dignity of Manner.* A medical man is a marked individual in a court room. A "don't care" attitude is not good. A frightened one gives a smart lawyer his chance, but a cool, calm demeanor with a "Be yourself" bearing makes for ease under most unusual and trying circumstances.

2. *Simplicity of Words.* This comes from the application of common sense born, possibly, of experience and the ability to stick to the truth at all times. One should "know his stuff" and "say his piece" simply but convincingly.

3. *Preparedness.* Preparedness means a complete history of the case in point, unaltered in wording, as made at the time of examination, not embellished by superfluous thoughts or unnecessary findings—a history truthful and reliable which will convince the Court that the testimony being given is right and for the purpose of getting at the truth in the case so that the correct decision may be reached by judge or jury.

4. *Explain: Do Not Argue.* Be explicit in answer to questions, intelligent in explanation of contested points, and kind in demeanor to the witness on the opposite side of the question. An argumentative attitude makes one a mark for sharp questioning and ridicule.

When you are right remember you can afford to keep your temper. When you are wrong you can't afford to lose it.

The medical witness can well keep in mind that dignity, simplicity of words, thorough preparedness and a non-argumentative bearing are the qualities best suited to inspire and command respect in Court as well as elsewhere.

Review From Russell Sage

The latest non-medical review of the medical situation in America is called "Physicians and Medical Care." It is a compact little volume prepared by Esther Lucile Brown of the Department of Statistics of the Russell Sage Foundation and published by the Foundation.

The book contains much universally accepted fact and just observation of course. It leads to a conclusion, however, which is worth quoting for the benefit of physicians because it is in agreement with the thought of most non-medical writers on the situation.

Says Miss Brown:

"If the problem of the nineteenth century was that of initiation of better medical schools and of concentration upon the basic sciences, the problem of the twentieth century is that of devising ways and means whereby adequate care can be provided for the entire population and physicians be compensated fairly for the service rendered. This is primarily an economic problem in which both physicians and society are involved, for both have much at stake. . . .

"Thinking Has Shifted"

"Various experiments such as those in the field of voluntary health insurance that have already been recorded, look toward a partial solution of a pressing need. Studies of compulsory health insurance and extension of state-supported medical services point to other ways of making care more widely available. It is evident that thinking has shifted in the direction of the needs of the masses and the demands of society become increasingly more exigent. Regardless of the attitude of those physicians and medical societies who would preserve the status quo, there are a great many individual physicians, members of health services and lay persons concerned with questions of health who believe that change is imminent and that only through change can there be progress.

A Call For Pioneers

"That serious difficulties confront those who seek to devise a means for extending medical care to the entire population while still preserving all that is best in medicine cannot be denied. . . . But the importance of finding a solution is commensurate with the difficulty involved. The challenge that confronts them has been well described by Dr. Henry Sigerist, who insists that American Medicine stands at the crossroads. By unremittingly purposeful effort it has equipped itself with hospitals, laboratories, schools and well-trained physicians. At present it is faced with the greatest task of all, that is, putting this artfully fashioned apparatus to work in such a way that all the people may receive medical care. 'It is an enormous problem, requiring a great deal of courage; for it is a question of treading on new ground, of going along untried paths. A new frontier has been opened, and calls for another generation of pioneers.'"

He Fears a Monkey Wrench

In this connection it is interesting to glance again over the mass of frank opinion from the physician pioneers themselves as it has been so effectively collected in the two volumes called "American Medicine," recently published by the American Foundation.

Here is one opinion, picked at random, that is to the point and typical of a considerable section of these published observations.

". . . Intelligent physicians are now seriously concerned and strenuously laboring over the economic problem and I think they should be allowed a decent opportunity to give their various experiments intelligent tryouts. Properly qualified medical groups have adequate brains, unlimited energy, and the finer motivation necessary to solve the problems that confront them. They are peculiarly well equipped for the effort and I am all for letting them have the field. . . .

"Of course, I may be unduly optimistic but I would rather maintain that frame of mind than to suffer the possible chagrin incident to the monkey wrench thrown into the machine by agencies less well equipped than is medicine herself to meet these problms. It will not be in the nature of a jeopardy of any sort, at this stage of the game, to follow the good old Italian counsel of *festina lente*. We Americans are all too prone, when things go badly, over a long enough period of time, to develop a frenetic energy that is bound up with the risk of making bad matters worse."

"A Good Fight"

From the *New York Medical Week:*

"Pennsylvania physicians are letting no grass grow under their feet in the fight against compulsory sickness insurance. Although President Roosevelt has invited the A. M. A. to help formulate a federal health policy in accord with professional principles, there is little doubt that an attempt will be made to amend the Social Security Act to include health insurance. Keystone State doctors are taking no chances!

"As in New York, 'On the Witness Stand' has been widely distributed to the laity to explain the fallacious theory and mischievous workings of compulsory health insurance. To the same end, 50,000 employers and workers have been circularized with a leaflet showing what obligatory sickness insurance would *cost them.* Physician-canvassers employed by the Pennsylvania Society, independent medical and allied professional organizations and individual practitioners are carrying on education activities along similar lines.

"To convince legislators of the force of popular sentiment against compulsory sickness insurance, 10,000 petition forms have been sent to physicians throughout

the state. Every practitioner has been urged to secure the signatures of five voters in his locality. Public opinion, mobilized in this fashion, rightfully exerts strong influence on legislative assemblies.

"The situation in Pennsylvania is essentially no different than in New York or any other state. Let compulsory health insurance get a foothold in one area, and it will spread like wildfire through the Union.

"In its lively campaign against obligatory sickness insurance the Pennsylvania profession is living up to the old adage that an ounce of prevention is better than a pound of cure. Its methods hold a valuable lesson for the profession because they can be employed by individual practitioners as well as organized groups."

Wisconsin Meeting

The Minnesota State Medical Association is in danger of losing its title, hitherto unchallenged, to the best state medical meeting in the United States.

Wisconsin, with its splendid 96th meeting, held in Milwaukee in September, is runner-up. Michigan meeting at Grand Rapids, September 27 to 30, is said to be planning the biggest meeting of its history.

The clear trend among state societies is toward a concentration of their energies in a large meeting with a special emphasis upon its values as a rallying point for all who are legitimately concerned in the care of the sick and with a special emphasis, also, upon its values for public health education.

The Wisconsin meeting was unique for its Hall of Health, a splendid collection of exhibits which the public was invited to visit. It was reliably estimated that more than 105,000 people visited the hall, studied exhibits on cancer, tuberculosis, heart and other conditions that depend upon a better public coöperation for their control. Scientific and technical exhibits were separately housed, and attracted, together with the distinguished program of lectures and round table discussions, a registration of some 1,850 physicians.

Minnesota men on the program included Dr. J. Arthur Myers, University of Minnesota; Dr. F. C. Rodda, Minneapolis; Dr. F. A. Willius, Rochester; Dr. Owen Wangensteen, Minneapolis; Dr. Edward T. Evans, Minneapolis. A fracture exhibit by Dr. M. S. Henderson, Dr. H. W. Meyerding, Dr. R. K. Ghormley and Dr. H. B. Macey of Rochester, was a feature, also, of the scientific exhibit section.

Dr. J. H. J. Upham, president, and Dr. Irving Abell, president-elect of the American Medical Association, and the distinguished medical writer Dr. Logan Clendening, all present as guest speakers, expressed themselves as enthusiastic about the meeting and particularly about the Hall of Health.

Pentobarbital Sodium-Abbott (Nembutal) Omitted from N.N.R.

The Council on Pharmacy and Chemistry reports that the Abott Laboratories has decided to market its brand of pentobarbital sodium under the name of "Nembutal." The Abbott Laboratories profess themselves entitled to the commercial monopoly of a proprietary name for this substance on the basis of discovery. They concede that they were not the discoverers of the free acid, but they contend that they discovered the novel therapeutic qualities and were the first to publish these. These qualities are nothing new in kind, for they are the same as those known for other similar barbiturates; the chief difference is that the action is briefer than that of some others, but this again is not fundamentally new, since it is merely a step in the gradation of action of the numerous barbiturate derivatives. However, it happened that, before the discovery was published by the Abbott Laboratories, it had also been made independently by another firm, Eli Lilly & Co.; and the only discovery of anything at all novel, the brief duration of action and its importance, did not originate from either of these firms but in the University of Wisconsin. It is true that the Abbott Laboratories published their work shortly before Eli Lilly & Co. It does not appear clear, however, that the public interest would be served by basing a monopoly merely on the date of publication. In view of these considerations, it appeared to the Council as definitely against the public interest to concede to the Abbott Laboratories, a monopolistic name for this product, which they did not invent, the chief actions of which were not new, and of which they had not been the sole investigators. The Council therefore gave to the substance the non-monopolistic name "Pentobarbital," based on its chemical characteristics; but in order to allow the Abbott Laboratories ample time for adjustment, it extended to them for a year the privilege of using the name "Nembutal," which they had copyrighted as a synonym. Later the Council extended the period for six months additional; but now that the period has expired, the firm informs the Council that it will return to the use of the copyrighted name. Under the circumstances, the Council can only withdraw its acceptance of the Abbott Laboratories brand of pentobarbital sodium, while continuing its acceptance of the Lilly brand. (J. A. M. A., August 14, 1937, p. 504.)

UNIVERSITY OF MINNESOTA—CENTER FOR CONTINUATION STUDY

PROGRAM ANNOUNCEMENT, 1937-1938

THE Center for Continuation Study of the University of Minnesota announces the program of medical seminars for 1937-1938. The faculty will be selected from the Medical School, Graduate School, Mayo Foundation, and General Extension Division, and will also include distinguished teachers from other medical centers. Lectures will be given in the class rooms of the Center, and clinics and demonstrations in the Medical School, University of Minnesota Hospitals, and affiliated institutions.

Each seminar will occupy the full time of the graduates from Monday to Saturday, inclusive. There will be no evening classes. Special library facilities for each seminar will be provided at the Center. If the interest warrants, lecture, clinic and demonstration mimeographed outlines will be sold for a nominal fee after each week's program. A special feature will be round table conferences at the close of the daily program to give the graduates an opportunity to ask questions.

Studies in Adult Education indicate that the most effective instruction is given by concentrating on limited fields. For this reason the program of each seminar will include only the subject material announced in advance. Also, the best results are apparently obtained from repeated exploration in a single field of knowledge with opportunities to apply this knowledge between seminars in the regular practice of the postgraduate students. Until this method is demonstrated to be inferior to courses of greater length the Unversity will continue to offer one-week seminars for physicians in active practice. Advanced classes will be formed for those seminar groups that desire it, provided the enrolment is sufficient and a suitable time for all concerned can be arranged.

Living Accommodations

The Center is located in the heart of the main campus of the University of Minnesota in Minneapolis. It is used exclusively for postgraduate instruction of men and women. Erected in 1936 at a cost of over $300,000, it is said to be the only educational unit of its kind in the United States. Containing living accommodations for 78 persons, it may be used simultaneously by several professional groups. Postgraduate physicians should plan on living in the dormitory of the Center. A double room with bath is $6.25 a week for each person; a single room without bath is the same price. Ample bathroom facilities on each floor are provided for those who select rooms without baths. Meals in the Center dining room are priced as follows: breakfast, 35 cents; luncheon, 45 cents; and dinner, 65 cents. Members of the physicians' families are welcome at the same rates. A large parking garage is in the basement; the daytime rate is 20 cents, and 24-hour parking is 50 cents.

Tuition

The tuition for each course is $25. This does not include living accommodations. In order to register for a seminar a fee of $3 should be sent in advance. This payment will be applied on the $25 tuition fee when registration is completed. In case the registrant fails to complete his registration the advance payment of $3 will be applied on the tuition for any future seminar. The right to cancel any course because of insufficient registration is reserved by the University, in which event the registration fee will be returned if desired.

Certificate

A certificate of attendance will be issued by the University of Minnesota upon recommendation of the chairman of the seminar committee and the director of the Center for Continuation Study.

Registration

Any licensed physician who is a member of his local or state medical association or of the American Medical Association may register for the seminars. Physicians residing outside the state are accepted on the same basis as Minnesota physicians. All physicians should register as far in advance as possible. This will give the chairmen of the seminar committees an opportunity to plan for the special needs of those who will attend. This planning has been an important factor in the success of the programs presented previously.

COURSES FOR MEDICAL GRADUATES

Clinics, Lectures and Demonstrations

Surgical Diagnosis and Treatment

November 1-6, 1937

A series of correlated presentations on the diagnosis and treatment of surgical conditions, mainly acute, but not including fractures and dislocations.

O. H. WANGENSTEEN, Professor and Head of the Department of Surgery, Medical School, and Associates.

Dermatology and Syphilology

December 6-11, 1937

The diagnosis and treatment of syphilis and the more prevalent diseases of the skin for those who encounter problems in these subjects in their practice.

HENRY E. MICHELSON, Professor and Director of the Division of Dermatology, Medical School, and Associates.

Ophthalmology and Otolaryngology

January 16-21, 1938

Studies of diseases of the eye, ear, nose and throat, including special conferences, the subjects to be selected by a survey of the prospective graduate students.

FRANK E. BURCH, Professor and Head of the Division of Ophthalmology; Horace Newhart, Professor and Director of the Division of Otology, Rhinology, and Laryngology, Medical School, and Associates.

Medical Diagnosis and Treatment

February 7-12, 1938

Diseases of the blood and blood-forming organs, gastrointestinal and respiratory tracts, and selected topics from other medical fields.

J. C. MCKINLEY, Head of the Department of Medicine, Medical School, and Associates.

Traumatic Surgery

March 7-12, 1938

The diagnosis and treatment of injuries, including fractures and dislocations.

WALLACE H. COLE, Professor and Director of the Division of Orthopedic Surgery, Medical School, and Associates.

Endocrinology

April 4-9, 1938

Present-day knowledge of the anatomy and physiology of the endocrine glands, clinical types of disorders, indications for and methods of treatment.

Committee from the preclinical and clinical departments of the Medical and Graduate Schools.

Diagnostic Radiology

Date to be announced (probably in June, 1938)

The technic and indications for the use of radiological methods in the diagnosis of disease.

LEO G. RIGLER, Professor and Head of the Department of Radiology, Medical School, and Associates.

Clinical Pathology

Date to be selected (at the convenience of the group)

A survey of the various fields of laboratory practice for clinical pathologists and others, in the technic and clinical interpretation of tests.

Committee from the Medical and Graduate Schools.

Proctology

Tentative

The diagnosis and treatment of disorders of the anorectal region.

WALTER A. FANSLER, Clinical Associate Professor of Surgery, Medical School, and Associates.

Other Seminars

Requests have been received for the formation of groups in addition to the seminars described above. To date, the number in each instance is not sufficiently great to form a class. If you are interested in any of the following subjects, will you please communicate with the director at your earliest convenience? Allergy, anesthesia, arthritis, deficiency diseases, degenerative diseases, diabetes, electrocardiography, gynecology, medical disorders of the kidney, liver and gallbladder, neurology, neurology and psychiatry, obstetrics, obstetrics and gynecology, orthopedics, pediatrics, physiotherapy, psychiatry, public health, refraction, surgical anatomy, minor surgery, surgical pathology, surgical technic, injection treatment of surgical diseases, and urology.

Information

Address all correspondence to the Director of the Center for Continuation Study, University of Minnesota, Minneapolis, or Dr.W illiam A. O'Brien, Medical Representative, at the same address.

OF GENERAL INTEREST

Dr. Carl G. Morlock, of Rochester, was recently married to Miss Katherine Mercer, also of Rochester.

* * *

Dr. W. Fetterly, of Minneapolis, has become associated with the Malmstrom-Sarff Clinic at Virginia.

* * *

Dr. E. H. Hansen, formerly of Minneapolis, has opened offices in Anoka, for the practice of medicine.

* * *

Dr. T. E. Broadie, superintendent of the Ancker Hospital, Saint Paul, was married September 7, 1937, to Miss Marjorie Allen of Attica, Indiana.

* * *

Dr. R. K. Proeschel, who was formerly associated with Dr. Sherwood of Kimball, has located in Willmar for the practice of medicine.

* * *

Dr. Kenneth Stein, of Lakeville, was married on August 24, to Miss Phyllis Boudin, of Lead, South Dakota.

* * *

Dr. A. F. Branton, of Willmar, was accepted as a member of the American College of Hospital Administrators, at the recent meeting of the American Hospital Association held in Atlantic City.

* * *

Dr. Hubert Lee, formerly of Northfield, has become associated with Dr. Nesmith Nelson of Brainerd, specializing in the treatment of eye, ear, nose and throat ailments.

* * *

Dr. E. H. Frost was chosen chief of staff of the Rice Memorial Hospital at Willmar, at a recent meeting. Dr. R. E. Anderson was selected as secretary of the association organized.

* * *

Dr. and Mrs. Roy F. Raiter, Cloquet, sailed on the steamer Aquitania on August 18 for Europe, where Dr. Raiter will spend two months attending various surgical clinics.

* * *

Dr. Arthur Neumaier of Durham, North Carolina, has joined the staff of the Raiter Hospital at Cloquet. Dr. Neumaier is a graduate of Duke University at Durham, North Carolina.

* * *

Dr. Peter Ward, superintendent of Miller Hospital, Saint Paul, was elected to the board of directors of the American Hospital Association, at the annual convention held recently at Atlantic City. He was chosen for a three-year term on the board.

* * *

Dr. Cecil A. Wilmot has become associated with his brother, Dr. H. E. Wilmot, of Litchfield, in the practice of medicine. During the past year and a half he has been located at Grand Rapids, where he was associated with Dr. McLeod.

* * *

Dr. Charles Sheppard, of Saint Paul, has become associated in the practice of medicine with his father and uncle, Dr. P. E. Sheppard and Dr. Fred Sheppard, of Hutchinson. Dr. Sheppard has recently completed his internship at Miller Hospital, Saint Paul.

* * *

Dr. A. E. Olson, of Duluth, has been appointed co-chairman of the committee on highway first aid service of the Duluth chapter of the American Red Cross, to direct the organization and maintenance of emergency highway first aid depots.

* * *

Dr. R. C. Hunt, of Fairmont, is contemplating the erection of a new hospital. Offices and consultation rooms will be located on the ground floor, and the second floor will be arranged to accommodate fifteen or more beds.

* * *

Dr. Myron O. Henry, of Minneapolis, was the guest speaker at the British Columbia Medical Association meeting held in Vancouver on September 13, 14 and 15. Dr. Henry presented papers on "Surgical Treatment of Fractures of the Hip" and "Spinal Fusion: Chip Graft Method."

* * *

Dr. Olaf Heiberg, resident physician at Minneapolis General Hospital, was married on September 4 to Miss Lois Shaffer, surgical superintendent at Minneapolis General Hospital. The wedding took place in Saint Cloud at the home of the bride's sister, Mrs. E. D. Surface.

* * *

Dr. J. E. Frank, of Springfield, has sold his practice to Dr. E. L. Penk of Stewart, Minnesota. Dr. Frank will open an office at Marshall. Dr. Penk is a graduate of the University of Minnesota medical school, and practiced at New England, North Dakota, before coming to Springfield.

* * *

Dr. Milton C. Rosenkrans has located at Wahpeton and has taken over the practice of the late Dr. Benjamin Thane. Dr. Rosenkrans was formerly located at Neillsville, Wisconsin. His wife, Sarah D. Rosenkrans, M.D., will remain in Neillsville temporarily to take care of the practice there.

* * *

Dr. W. W. Yaeger, of Ivanhoe, has taken over the practice of Dr. L. J. Happe, at Marshall. Dr. Yaeger is a graduate of the University of Minnesota medical school, and practiced surgery in South Dakota for two years. He has practised at Ivanhoe for the past nine years.

Dr. M. M. Loucks has become associated with Dr. A. G. Chadbourn of Heron Lake. He was formerly at the station hospital at Fort Cook, Nebraska. Dr. Loucks is a graduate of the University of Minnesota medical school and was a member of the faculty for six years. He was in charge of the x-ray and laboratory departments at the army hospital at Fort Cook.

* * *

Physicians of twenty-four states and of the Philippine Islands are receiving reprints of "The Doctor and Tuberculosis of the Future," the third annual Dr. John W. Bell Tuberculosis Lecture before the Hennepin County Medical Society delivered last December by Dr. H. E. Kleinschmidt, Medical Director of the National Tuberculosis Association.

Reprints of the published lecture, as it appeared in MINNESOTA MEDICINE, have been purchased by tuberculosis associations for distribution to physicians in Colorado, Georgia, Indiana, Iowa, Kansas, Kentucky, Louisiana, Maryland, Michigan, Minnesota, Mississippi, Nebraska, New Hampshire, New Jersey, North Carolina, Oregon, Pennsylvania, Texas, Utah, Vermont, Washington, Brooklyn, New York city, Chicago and Hawaii and Kauai, in the Philippine Islands.

It is a matter of gratification to the Hennepin County Medical Society that this lecture, locally sponsored, is receiving such widespread attention. The Lectureship was established in the Society three years ago by the Hennepin County Tuberculosis Association, which has not yet announced the name of an outstanding leader in the field of tuberculosis who will deliver the lecture during the current year.

* * *

Dr. Walter J. Marcley, of Minneapolis, for the past 30 years active in tuberculosis prevention work, was the guest of honor at a dinner September 28, given by fellow workers and friends on the occasion of his resignation as chief of the Tuberculosis Service of the United States Veterans' Hospital. Dr. Marcley was medical director of the first state sanatorium for the treatment of tuberculosis in the United States at Rutland, Vermont. He was one of the founders of the National Tuberculosis Association in 1904. In 1907, he returned to his native state to act as medical director of the Minnesota State Sanatorium at Walker.

During the World War, he was a member of the Rockefeller Tuberculosis Commission and he also served as Chief Physician to the American Red Cross, in Switzerland. Dr. Marcley is a past president of the Minnesota Public Health Association, the Mississippi Valley Conference, and of the Hennepin County Tuberculosis Association. He will give his full time to his private practice.

Causalin (Causyth) Not Acceptable for N.N.R.

The Council on Pharmacy and Chemistry reports that "Causalin" is another name for Causyth, which was exploited several years ago in Austria as a treatment for arthritis. A numer of recent inquiries from American physicians about Causalin prompted a review of the present claims for the product. The status of Causalin is about the same as that of Causyth reported in *The Journal A. M. A.*, March 1, 1930. At that time a referee, who reviewed most of the reports on Causyth in German medical literature, held the evidence unsatisfactory and uncritical and the product therapeutically worthless and ineffective, except for possible shock effects which might be dangerous. Today the product is being exploited as Causalin in this country by the Amfre Drug Co., Inc., 31 East Twenty-Seventh Street, New York City. Presumably the product is manufactured abroad and distributed by this agency in this country. Clearly, there is nothing original about the product, consisting as it does of the well known official drug aminopyrine (U.S.P.) and the active chemical group hydroxyquinoline or chiniofon (U.S.P.) which is an amebicide and a mild wound antiseptic. Quinoline is also an important chemical group in quinine (U.S.P.) and cinchophen (N.F.). Accordingly, such statements as the following in the advertising literature appear misleading: "Causalin (aminodimethyl-pyrazolon - quinoline - sulphonate) contains no cinchophen and no cinchophen derivatives." The claims for Causalin (Causyth) are exaggerated, misleading and unwarranted, and the product has no place in the therapeutic armamentarium of responsible, qualified physicians. The exploiters of the product are to be censured for omissions of warning concerning its dangerous toxic potentialities, for obscuring the essential composition of the product by using an unnecessarily long and unfamiliar chemical name for the well known drug aminopyrine, and for using the noninformative name Causalin. There is no good reason to believe that the claimed composition of Causalin entitles it to be considered a chemical compound as indicated in the advertising; apparently it is a mixture, without originality or rationality. Causalin is to be condemned as an unsafe and dangerous product, the exploitation of which is against the interest of the public and the medical profession. The Council declared Causalin (Causyth) not acceptable for N.N.R. for the reasons given in the foregoing report. (J. A. M. A., August 14, 1937, p. 506.)

Dosage of Preparations Containing Vitamins A and D

The Council on Pharmacy and Chemistry held that the recommended dosages for capsule and tablet preparations of vitamins A and/or D should not be less than the minimum dosage for infants and adults equivalent to two teaspoonfuls of cod liver oil—U.S.P. (minimum strength). The Council voted the requirement (1) that the dosages of accepted preparations of vitamins A and/or D provide at least the equivalent in these vitamins of two teaspoonfuls of cod liver oil but not more than 10,000 units of vitamin A and 1,000 units of vitamin D, and (2) that dosage statements on labels and in advertising should be accompanied by the phrase "or as prescribed by your physician." (J. A. M. A., August 14, 1937, p. 507.)

REPORTS and ANNOUNCEMENTS

MEDICAL BROADCAST FOR OCTOBER

The Minnesota State Medical Association Morning Health Service

The Minnesota State Medical Association broadcasts weekly at 9:45 o'clock every Saturday morning over Station WCCO, Minneapolis and Saint Paul (810 kilocycles or 370.2 meters).

Speaker: William A. O'Brien, M.D., Associate Professor of Pathology and Preventive Medicine, Medical School, University of Minnesota. The program for the month will be as follows:

October 2—Heart Disease.
October 9—Hand Infections.
October 16—Dietary Dangers.
October 23—Hemorrhage.
October 30—Dental Health Education.

MAYO FOUNDATION LECTURES

A special program of lectures and demonstrations in surgery and medicine will be held under the direction of The Mayo Foundation from November 8 to 12, inclusive. Mornings will be devoted to surgical and medical clinics. In the afternoons and evenings, in addition to a clinico-pathologic conference, symposiums will be conducted on gastro-enterology, sulfanilimide therapy, hematology, neurology, allergy, diseases of the chest and cardio-vascular diseases. Visiting physicians are invited to attend.

MINNESOTA STATE ASSOCIATION MEETING

The 85th Annual Meeting of the Minnesota State Medical Association will be held June 29 and 30 and July 1, 1938, at the Hotel Duluth in Duluth.

Dr. J. M. Hayes, president-elect of the Association, is general chairman of the committee in charge of program arrangements. The surgical section of the committee includes Dr. Stanley Maxeiner, Minneapolis, chairman, Dr. V. S. Counseller, Rochester, secretary, and Dr. Roscoe Hunt, Fairmont. Dr. P. G. Boman, Duluth, is chairman of the medical section; Dr. D. B. Souster, Saint Paul, secretary, and Dr. J. N. Libert, St. Cloud. Dr. R. J. Moe, Duluth, is general chairman of local arrangements. Dr. W. A. O'Brien is chairman of the section on specialties.

All members who wish to present papers or exhibits at the meeting are urged by the committee to send their requests promptly to state headquarters.

EAST CENTRAL MINNESOTA MEDICAL SOCIETY

The East Central Minnesota Medical Society met at Braham, Minnesota, on September 22, 1937. Dr. Elton Clothier of Elk River was elected to membership in the society. Dinner was served and in the evening a scientific program was presented by guest speakers. Dr. T. A. Peppard of Minneapolis spoke on the "Use of Digitalis and Quinidine in Heart Diseases" and Dr. Otto Yoerg, also of Minneapolis, gave an address on "Improved Treatment of Fractures of Os Calcis." Both addresses were supplemented by moving pictures and slides.

WABASHA COUNTY MEDICAL SOCIETY

The sixty-ninth annual meeting of the Wabasha County Medical Society will be held at Kellogg, Minnesota, on Thursday, October 7, in the parlors of the Methodist Episcopal Church. The business session will be held at 4 P. M., and the scientific session at 7:30 P. M. The following program has been arranged:

"Review of the Methods of the Induction of Fever for Therapeutic Purposes"—Dr. B. A. Flesche, Lake City.

"The Use of Sulfanilamide"—Dr. E. G. Bannick, Mayo Clinic, Rochester.

"Tuberculosis in Southeastern Minnesota"—Dr. R. H. Frost, Wabasha.

"Obstetric Problems; Anesthesia and Analgesia in the Home"—Dr. G. E. Hudson, Minneapolis.

"The Medical Treatment of Diseases of the Gall Bladder"—Dr. J. F. Weir, Mayo Clinic, Rochester.

Dr. B. A. Flesche of Lake City is president of the Society, Dr. J. S. Collins of Wabasha is vice president, and Dr. W. F. Wilson of Lake City is secretary.

NORTHERN MINNESOTA MEDICAL ASSOCIATION

The seventeenth annual meeting of the Northern Minnesota Medical Association was held at Virginia, Minnesota, August 27 and 28. The following officers were elected for the coming year:

President.....Dr. J. F. Norman, Crookston
Vice President.....Dr. O. W. Parker, Ely
Secretary-Treasurer.....Dr. Clarence Jacobson, Chisholm

Crookston was named next year's meeting place of the Association.

Dr. Frank Hirschboeck, of Duluth, served as toastmaster at the banquet Friday evening. Professor J. A. Merrill, president emeritus of the Superior State Teachers College, was the guest speaker. His subject was "The Wonderland of Lake Superior." He described the land in Northern Minnesota and upper Wisconsin and Michigan as the "eighth wonder of the world," stating that this area is the oldest place, from a geological standpoint, in America, if not in the world. He explained that a motorist in this territory could in twenty-four hours witness earth formations which took nature two billion years to build. Among the wonders named were Jasper Peak north of Ely, the iron mines, the volcanic ridge north and south of

Lake Superior, the copper mines of Michigan, and Lake Itasca, source of the Mississippi.

Dr. Leland of the American Medical Association staff gave an address on the "Business Side of Medicine." Dr. A. W. Adson, president of the Minnesota State Medical Association, in his address asked the doctors to offer encouragement to the young men in the profession and to give them a place in the work.

In his annual address, Dr. O. O. Larsen appealed to the physicians to increase their participation in the national crusade to eliminate venereal disease.

The Side Actions of Barbitals

Barbituric acid and the various compounds derived from it by alteration of the molecule are now widely used. Extensive employment by qualified practitioners and also by the public has grown to proportions that indicate lack of knowledge of the side actions of these drugs. Even more blameworthy is the practice of introducing new and more potent barbitals without reliable investigation of their pharmacologic actions. Hanzlik indicates three experiments which illustrate depression and paralysis of peripheral neuromuscular elements in the autonomic system caused by amytal, one of the highly active barbitals, which has been promoted for intravenous analgesia or anesthesia. The chain of evidence on the side actions of the arbitals justifies the conclusion that ganglionic paralysis or synaptic block in the cardiac vagi may follow the intravenous or intraperitoneal administration of amytal, an action paralleled commonly by such poisons as nicotine and lobeline and sometimes by choline. On the other hand the ganglionic paralysis, together with the central depression of barbital, would make an effective combination for controlling asthmatic attacks of reflex origin, a point that is worthy of further investigation and that again illustrates the value of detailed pharmacologic consideration. All in all, these side actions emphasize that all the barbitals cause widespread depression or paralysis of living tissues in varying degrees and that there is no such thing as a nontoxic sedative or anesthetic that may be used with impunity. Some of the side actions might easily obscure or confuse a diagnosis and affect a patient's recovery from a disease in which the barbital was exhibited merely as a symptomatic or palliative measure. The general condition of the patient should always be kept in mind when barbitals are administered. A number of state legislatures have passed or are in the process of passing measures limiting the sale of barbitals to prescriptions signed by physicians, dentists or veterinarians. Even better than legislation in this respect is an education of barbital users to the attending dangers of these compounds. Like most drugs, they are two-edged swords, useful in the hands of those competent to administer them, dangerous in the hands of the incompetent. Additional special knowledge, bearing particularly on the pharmacologic characteristics of various hypnotics and on the clinical evaluation of the patient, is essential (J. A. M. A., August 14, 1937, p. 508.)

WOMAN'S AUXILIARY

MRS. J. F. NORMAN, Crookston, *President*
MRS. A. A. PASSER, Olivia, *Editor*

The Woman's Auxiliary mourns the death of Mrs. Stephen Baxter—who died unexpectedly, July 29, while on a visit at the home of her son-in-law and daughter, Captain and Mrs. Benjamin Eaton Thurston (Betty Gay Baxter), at West Point. Deepest sympathy is extended to her husband, Dr. Stephen H. Baxter, and the other surviving relatives.

Mrs. Baxter was president of the Woman's Auxiliary of the Hennepin County Medical Association at the time of her death and for many years had taken an active part in numerous other organizations.

Laura Robb Baxter possessed many admirable attributes and was beloved generally. She leaves a host of friends who mourn deeply her untimely death.

Board Meeting

The fall meeting of the Executive Board was held at the Town and Country club, Saint Paul, Tuesday, October 5, at 10 A. M.

Mrs. J. F. Norman, Crookston, state president, presided at the meeting, and various matters of importance were discussed. Reports of chairmen and county presidents were read. Following the luncheon the board members were taken to the Coach adjoining the Medical Association offices at 11 West Summit Avenue, where short addresses were given by Dr. James Hayes, Minneapolis, Doctor L. R. Critchfield and Dr. Robert Burns of Saint Paul.

PRESIDENT'S ADDRESS*

MRS. J. F. NORMAN, *Crookston, Minnesota*

Madame Chairman, Mrs. Blake, Members of the Auxiliary and Guests:

Our State Auxiliary was organized in October, 1922. We are therefore entering upon our sixteenth year as a state organization.

If we may compare the life of an organization to that of individuals, the thought occurs that at no past or future time in their lives do hope, and the will to achieve, run quite so high as during that sixteenth year. The age accepts no discouragement, admits no defeat, but looks ahead with confidence and a determination to win over any obstacle which life may present.

But hand in hand with high courage at this time in the life of individuals, we occasionally discover an intolerance of things as they are, and an impatience to bring about a new order in the least possible time, together with the feeling that everything which the past has to offer has already been learned.

*Presented before the Woman's Auxiliary of the Minnesota State Medical Association at the annual meeting, Saint Paul, Minnesota, May 4, 1937.

As an organization, then, let us hold fast to the virtues and avoid the pitfalls which accompany the age we are entering. Let us, individually and collectively, realize and appreciate the work which has been accomplished by our splendid leaders, state and national, during these past years, and remind ourselves that we owe a debt of gratitude to all those who have given unreservedly of their talents and their time to build this organization which we now enjoy.

As members of the Auxiliary it is well to remember the confidence reposed in us by the leaders of the State Medical Association, our parent organization — those leaders who have told us that the most potent forces in overcoming unethical efforts to treat the sick, must, and will be, the State and County Auxiliaries.

How, then, can we qualify for this work and make ourselves deserving of this confidence? By becoming well-informed members of our local Auxiliary. Not by feeling that it is only the officers in each group who must know what is happening in the world of medical economics and the problems confronting organized medicine, but by accepting the responsibility for ourselves, individually, as members of the group.

Becoming informed is a continuous process. It means reading the Organization Section of the *Journal of the American Medical Association,* which includes news of the Woman's Auxiliary; reading the Medical Economics section and State Auxiliary news in MINNESOTA MEDICINE; reading *Everybody's Health* and *Hygeia;* giving attention to the radio broadcasts on health and knowing what legislation the Medical Association sponsors; and it means subscribing to and becoming familiar with the material given in the News Letter which is sent out by the National Auxiliary. The News Letter has become a vital force in the life of the Auxiliary, and is an interesting medium of information.

Does such a program sound formidable? Not necessarily, because it all takes less time than the list indicates; and, after her home, what should interest the doctor's wife to a greater extent than the welfare of the profession which her husband serves? Through information thus acquired she becomes a participant in public relations, as a member of other organized groups. Through her tactful direction, these groups are brought to the medical profession for speakers and for authentic material on everything pertaining to health.

But frequently in the minds of Auxiliary members there is the thought that any effort they make in the service of their Auxiliary will be misconstrued by the public, and they will be accused of attempting to further the personal interests of their husbands. If it can be kept in mind that all auxiliaries are organized to serve the interests of organized medicine as a whole, especially for the purpose of promoting good fellowship within the profession and assisting in the work of health education, the public will be quick to understand that members of the auxiliary are not using the organization to promote the interests of the individual doctor, but are merely assisting organized medicine in its self appointed task of guarding the health of each community.

In the beginning, the Auxiliary existed almost entirely for the purpose of handling the social side of conventions, which in itself requires capable organization and management. But, since women are constituted as they are, with a feeling that no organization has the right to exist merely for the pleasure of its members, the various County and State Auxiliaries soon turned their attention to work which seemed to be waiting, and could be best accomplished by women who have the privilege of expert guidance from the medical profession. The results are apparent in the splendid reports heard at the annual meeting today.

You have noted that foremost in the minds of Auxiliary members are matters pertaining to the health and welfare of individuals and communities, and health education in various forms receives the greatest share of attention. This is as it should be, for one of the objectives of the Auxiliary is to pass on to the public the point of view of the medical profession, and keep health leadership where it belongs, with the profession.

Many communities are carrying on this work through public health agencies, and these agencies welcome the assistance and coöperation of the doctor's wife. In aiding them she is promoting the interests of the profession, to the end that organized medicine and not organized lay opinion, interprets the medical arts to the public.

We hear it stated that health education has received so much attention that it is already well in hand and will go forward successfully with or without our assistance. Fortunately, this is true, but it is work which has no ending as long as the human race endures. While young people and old continue to suffer from tuberculosis, while life is cut short by cancer, while smallpox and diphtheria still take their toll, and while the rickets of childhood and anemia and malnutrition in young women continue to be serious factors in the maternal death rate, there is work enough in health education to engage the attention of all of us.

During the coming year, it will be my earnest desire to be of service to each Auxiliary, and with the friendly spirit and loyalty to the State Organization which exists within each group, we shall hope to experience continued growth and progress during this sixteenth year of our existence.

If the (966) women of the Minnesota Medical Auxiliary will give their informed and intelligent consideration to the special problems which are the concern of the medical profession at the present time, they can be a powerful, unobtrusive force in the final solving of those problems.

BOOK REVIEWS

Books listed here become the property of the Ramsey and Hennepin County Medical libraries when reviewed. Members, however, are urged to write reviews of any or every recent book which may be of interest to physicians.

SYPHILIS, THE NEXT GREAT PLAGUE TO GO. Morris Fishbein, M.D., editor of *Journal of the American Medical Association* and *Hygeia*. 70 pages. Illus. Price, $1.00, cloth. Philadelphia: David McKay Co., 1937.

PSYCHIATRIC NURSING. William S. Sadler, M.D. Chief Psychiatrist and Director Chicago Institute of Research and Diagnosis; Consulting Psychiatrist to Columbus Hospital. In collaboration with Lena K. Sadler, M.D., and Anna B. Kellogg, R.N. 433 pages. Price, $2.75, cloth. St. Louis: C. V. Mosby Co., 1937.

THE TRAFFIC IN HEALTH. Charles Solomon, Assistant Clinical Professor of Medicine, Long Island College of Medicine; Lecturer in Materia Medica, Training School for Nurses, Jewish Hospital of Brooklyn. 393 pages. Price, $2.75, cloth. New York: Navarre Publishing Co., 1937.

EMOTIONAL ADJUSTMENT IN MARRIAGE. Le Mon Clark, M.S., M.D., Assistant in Obstetrics and Gynecology, University of Illinois College of Medicine, 261 pages. Price, cloth, $3.00. St. Louis: C. V. Mosby Co., 1937.

YOUR DIET AND YOUR HEALTH. Morris Fishbein, M.D. Editor of the *Journal of the American Medical Association; Hygeia,* the Health Magazine. 298 pages. Price, cloth, $2.50. New York: Whittlesey House, McGraw-Hill Book Co., 1937.

SYNOPSIS OF GYNECOLOGY, based on the textbook *Desires of Women.* By H. S. Crossen and R. J. Crossen. 2nd Edition, 247 pp. Price $3.00. St. Louis: The C. V. Mosby Company, 1937.

This synopsis well covers the field for which it is intended, *i.e.,* for those who need to know this subject only in a general way. It is concise, covers practically all points necessary in the general course, and does not overload the student with too detailed descriptions of pathologic conditions. The figures illustrate the text in a satisfactory manner. It is a valuable contribution to textbooks on the subject.

H. M. WYNNE, M.D.

PHYSICAL DIAGNOSIS. Don C. Sutton, M.S., M.D., Associate Professor of Medicine, Northwestern University School of Medicine. 495 pages. Illus. Price, $5.00. St. Louis: C. V. Mosby Co., 1937.

Physical Diagnosis by Sutton is a well-organized work covering all the phases of physical diagnosis, in a concise and very readable style. The book should be especially valuable to the medical student. The emphasis placed on the use of the senses in diagnosis is timely, for clinical observation in the care of the patient is still important, laboratory data subordinate.

An Historical Introduction by Irving S. Cutter is a most valuable addition. The volume closes with an excellent, comprehensive index.

It seems unfortunate that such a generally excellent volume should be handicapped by the use of a glossy, glaring paper which makes reading difficult, especially under artificial light.

H. O.

SYNOPSIS OF DIGESTIVE DISEASES. By John L. Kantor, Ph.D., M.D., Associate in Medicine, Columbia University. 300 pages. Illus. Price $3.50. St. Louis: C. V. Mosby Co., 1937.

This little book is what it purports to be, a synopsis. The author, who is known to us as the author of "The Treatment of Common Disorders of Digestion," published in 1924, succeeds in condensing a great deal of material into small space. For the most part the essential facts concerning the digestive diseases are well and accurately stated and, while it would be too much to ask to get any two men to agree entirely upon certain details, there is very little to call forth any adverse criticism. The author has had adequate experience to qualify him as a gastro-enterologist, he being an associate in medicine at Columbia University and gastro-enterologist at the Montefiore Hospital. The book is illustrated and is a very satisfactory synopsis.

T. A. PEPPARD, M.D.

ANNUAL REPRINTS OF THE REPORTS OF THE COUNCIL ON PHARMACY AND CHEMISTRY of the American Medical Association for 1936, with the Comments That Have Appeared in the Journal. Cloth. Price, $1. Pp. 104. Chicago: American Medical Association, 1937.

This book is essentially a record of the negative actions of that distinguished body, the Council on Pharmacy and Chemistry of the American Medical Association; that is, it sets forth the findings concerning medicinal preparations which the Council has voted to be unacceptable for recognition and use by the medical profession. Many of the reports record outright rejection or the rescinding of previous acceptances; others report in a preliminary way on products which appear to have promise but are not yet sufficiently tested or controlled to be ready for general use by the profession.

Among the reports on out-and-out unacceptable products are Amend's Solution and the "Igol" products, iodine preparations marketed under misleading or unacceptable claims, the latter under an uninformative proprietary name; Androstine-Ciba, claimed to be a testicular extract and found to be an irrational combination of inactive preparations, marketed with unwarranted and misleading claims; Gadoment, a preparation of cod liver oil in a wax base with zinc oxide benzoin and phenol, proposed for use in the treatment of burns, cuts and minor skin irritations, found unacceptable as being an unoriginal product of insufficiently declared

composition marketed under a coined proprietary name with unwarranted therapeutic claims, and indirectly advertised to the public; the "Carasyl" preparations, which are essentially mixtures of psyllium flour, karaya gum and fig flour, marketed with unsubstantiated therapeutic claims under a proprietary name.

In 1934 the Council sponsored an exhaustive report on bacteriophage therapy which pointed out that in view of the present status of knowledge, no such preparations could be accepted for New and Non-official Remedies. In this volume of the collected Council reports, the Council declares the "Phagoid" preparations, a line of bacteriophage products, definitely unacceptable because they are offered to the medical profession with unscientific, unwarranted claims, thus encouraging physicians to use in a routine way medicaments, the therapeutic value of which had not been established, and because the preparations conflicted in other ways with the rules of the Council.

This volume includes a preliminary report on Trichophytin and Oidiomycin-trichophyton preparations marketed by Lederle Laboratories, Inc. This report is a sequel to the preliminary report on Trichophyton Extract issued in 1932, which postponed consideration to await development of further clinical evidence on Trichophyton therapy. Also included in this volume is a report on the unacceptability of two trichophyton preparations, Dermatomycol and Dermotricofitin, distributed in this country by Ernst Bischoff Co., Inc., under the stated proprietary names without sufficiently declared composition and with unwarranted therapeutic claims.

Other preliminary reports are Refined and Concentrated Antipneumococcic Serum Type VII-Lederle, Present Status of Tetrachlorethylene (since accepted for N.N.R.), Smallpox Vaccine (from Chick Chorio-Allantoic Membrane)-Lilly, and Use of Trichloroethylene for General Anesthesia.

CLASSIFIED ADVERTISING

WANTED: Young physician to take care of my practice in North Dakota for a month or six weeks. Give references, state salary expected, your age, qualifications, and other particulars in first letter. To begin work here about first week in November. Address D-385, care MINNESOTA MEDICINE.

FOR SALE: Complete office outfit, furniture, surgical, obstetric, electrical, nose and throat instruments, medical and surgical books. Priced very low for cash or endorsed note. Owner disabled. Address D-377, care MINNESOTA MEDICINE.

WANTED—Doctor to share office in Medical Arts Building, Minneapolis. Address D-383, care MINNESOTA MEDICINE.

WANTED: Position as secretary and technical assistant in doctor's office by young woman with some experience, excellent health, and good educational and social background. Opportunity for growth and experience more important than initial salary. References upon request. Address D-384, care MINNESOTA MEDICINE.

WANTED—Doctor, middle aged. Good progressive town. Population 500. No doctor within radius of 20 miles. Money maker for right man. Investigate. Write Lawler Drug Store, Conde, S. D.

EXCELLENT OPPORTUNITY for a doctor in a town of 2,300. Write A. B. Hannes, Harvey, North Dakota.

LABORATORY TECHNICIAN desires position. Hospital experience. Address Erna Miller, 2820 Riverside Avenue South, Minneapolis, Minn.

PRACTICE FOR SALE — Fully equipped office, x-ray, fluoroscope, basal metabolism, etc. $2,000. Terms if desired. Specializing. Percentage and guarantee with privilege of buying later if desired. Address C. P. Truog, Lindstrom, Minn.

CO-TENANT WANTED

To share with one other physician the space, equipment and technical service of his present completely furnished and equipped office in the Lowry Medical Arts Building, St. Paul.

A BARGAIN IN OFFICE SPACE, FACILITIES AND SERVICE

$100.00 per month includes:

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FOR FURTHER INFORMATION ADDRESS

Frederic E. B. Foley, M.D.
229 Lowry Building
Saint Paul, Minnesota

LOCUM TENENS wanted the latter part of October. Address D-386, care MINNESOTA MEDICINE.

WANTED: Dentist—large unopposed territory. Good income from the start. Modern brick office suite with physician. Main line Great Northern Railway. W. E. Wray, M.D., Campbell, Minnesota.

FOR SALE—Have practiced over 40 years. Wish to retire. Located East Central Minn. Good roads and good practice. Good for five thousand per year, good collections. Can turn over county work in two counties. Small town. Good house and office. Will sell cheap. Address, Lock Box No. 1, Finlayson, Minn.